

- **Clinical Importance:**

- Early detection and appropriate treatment are crucial as CNS tuberculomas can lead to significant neurological deficits.
- Differential diagnosis should be made with other space-occupying lesions in the brain, as treatment strategies differ.

- **Diagnosis:**

- Imaging techniques like CECT and MRI play a pivotal role in diagnosing tuberculoma.
- The radiological findings, when correlated with clinical symptoms and history of TB exposure or infection, can assist in establishing a definitive diagnosis.
- Sometimes, a biopsy might be required, especially when imaging is inconclusive and the clinical suspicion remains high.

- **Differential Diagnosis:**

- It's vital to differentiate tuberculomas from other CNS lesions that might present similarly on imaging. Conditions such as cryptococcal meningitis, CMV encephalitis, toxoplasmosis, sarcoidosis, meningeal metastases, and lymphomas can have overlapping radiological findings.

Differential Diagnosis	Key Features	Imaging Findings	Additional Notes
CNS Tuberculoma	Headache, seizures, focal neurological deficits	Ring-enhancing lesions, surrounding edema, calcifications in chronic cases	Associated with TB infection, immunocompromised state
Neurocysticercosis	Seizures, headache, hydrocephalus, sometimes asymptomatic	Multiple cystic lesions of varying stages, ring-enhancing lesions, calcifications	Caused by Taenia solium (pork tapeworm)
Brain Abscess	Fever, headache, focal neurological	Ring-enhancing lesion with surrounding	Often associated with bacterial infection

	deficits, altered mental status	edema, central necrosis	
<i>Glioma (Brain Tumor)</i>	Headache, seizures, progressive neurological deficits	Mass effect, variable enhancement, may have necrosis	Primary brain tumor, can be benign or malignant
<i>Metastatic Brain Lesions</i>	History of primary cancer elsewhere, neurological deficits	Multiple lesions, ring-enhancing, variable edema	Secondary brain tumors from cancer spread
<i>Toxoplasmosis (in Immunocompromised)</i>	Fever, headache, seizures, focal deficits	Multiple ring-enhancing lesions, often in basal ganglia	Caused by <i>Toxoplasma gondii</i> , common in HIV/AIDS
<i>Primary CNS Lymphoma</i>	Altered mental status, focal deficits, seizures	Homogeneously enhancing mass, periventricular location	More common in immunocompromised individuals
<i>Multiple Sclerosis</i>	Visual disturbances, motor/sensory deficits, relapsing-remitting course	Multiple white matter lesions, Dawson's fingers	Autoimmune demyelinating disorder
<i>Vascular Malformations</i>	Headache, seizures, possible hemorrhage	Abnormal vascular structures, possible hemorrhage	Includes arteriovenous malformations, cavernomas

- **Treatment:**
 - Antituberculous therapy (ATT) remains the mainstay of treatment.

- In some cases, surgical intervention might be required, especially in large lesions causing significant mass effect or those leading to obstructive hydrocephalus.
- **Prognosis:**
 - With timely and appropriate treatment, the prognosis of CNS tuberculoma is generally favorable.
 - It's essential to monitor patients for potential complications and ensure adherence to antituberculous medication to prevent recurrence or worsening of the disease.

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Q: Differences between tuberculoma and NCC

Feature	CNS Tuberculoma	Neurocysticercosis
Causative Agent	Mycobacterium tuberculosis	Taenia solium (pork tapeworm)
Epidemiology	More common in regions with high TB prevalence	Endemic in areas with poor sanitation and where pigs are raised
Risk Factors	TB infection, immunocompromised state	Ingestion of food/water contaminated with tapeworm eggs, poor hygiene
Clinical Presentation	Headache, seizures, focal neurological deficits	Seizures, headache, hydrocephalus, sometimes asymptomatic
CSF Analysis	Lymphocytic pleocytosis, elevated protein, low glucose	Eosinophilia (rare), elevated protein, normal or mild lymphocytic pleocytosis
Serology	Not typically useful for diagnosis	ELISA for cysticercosis antibodies
Treatment	Antituberculous therapy, corticosteroids	Antiparasitic drugs (albendazole or praziquantel), corticosteroids, antiepileptic drugs
Prognosis	Generally good with treatment, but can be fatal if untreated or in severe cases	Variable, depends on the number, size, and location of cysts; can be fatal if untreated

Radiological differences:

Feature	CNS Tuberculoma	Neurocysticercosis
CT Scan Appearance	Well-defined, round/lobulated lesions; Ring or nodular enhancement; Calcification in chronic stage; Surrounding edema	Stages: vesicular, colloidal, granular-nodular, calcified; Vesicular: cystic lesions, scolex visible; Colloidal: ring-enhancing lesions, edema; Granular-nodular: nodular/ring-enhancing, less edema; Calcified: calcified lesions
MRI Findings	T1: hypointense/isointense; T2: hyperintense with hypointense rim; Post-contrast: ring/nodular enhancement; DWI: restricted diffusion possible	Vesicular: isointense to CSF, scolex as mural nodule; Colloidal: hyperintense on T1, hypointense on T2, ring enhancement; Granular-nodular: nodular enhancement; Calcified: hypointense on all sequences; DWI: restricted diffusion in colloidal stage
Advanced Imaging	MRS: lipid peaks, decreased NAA; Variable perfusion patterns	MRS: elevated alanine peak in vesicular stage
Enhancement Pattern	Ring or nodular enhancement	Ring enhancement in colloidal stage; nodular in granular-nodular stage
Calcification	Common in chronic stage	Present in calcified stage
Edema	Surrounding edema common	Pronounced in colloidal stage; less in granular-nodular stage
Characteristic Features	Consistent appearance; may involve basal ganglia and meninges	Distinct stages; presence of scolex; random distribution of lesions

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Q: Role of Steroids in TB

Steroids are a valuable adjunctive therapy in certain forms of TB where inflammation significantly contributes to morbidity. Their use should be guided by clinical indications, and patients should be closely monitored for potential side effects.

The goal is to balance the benefits of reducing inflammation and alleviating symptoms with the risks associated with steroid therapy.

- **Immunological Response:** Interaction between microbial factors and host immunological factors can lead to the release of pro-inflammatory markers like IL-2 and Interferon-gamma. This can cause a paradoxical worsening of symptoms in TB.
- **Paradoxical Reaction:** Majority of patients recover with the continuation of anti-TB therapy. However, some may experience severe, life-threatening manifestations and sequelae.
- **Inflammation Suppression:** In cases where inflammation significantly contributes to the disease severity, steroids can be beneficial in suppressing the inflammatory response and providing symptomatic relief.

Indications for Steroid Therapy in TB

- **Tuberculous Meningitis (TBM):** All children with TBM should be treated with adjuvant steroids, regardless of disease severity.
- **Pericarditis:** In TB-related pericarditis, steroids can reduce inflammation and prevent constrictive complications.
- **Addison's Disease:** In TB-related adrenal insufficiency, steroids are essential for management.
- **Miliary TB with Alveolo-Capillary Block:** Steroids can help in managing severe respiratory distress.
- **TB Uveitis:** Steroids are used to reduce ocular inflammation.
- **Other Intracranial TB:** The evidence for the use of steroids in intracranial TB like tuberculomas is unclear but may be considered in severe cases.
- **Endobronchial TB, Bronchial Compression, Mediastinal Compression Syndrome:** Steroids may be used to reduce compression and inflammation.
- **Pleurisy with Severe Distress:** To alleviate severe pleuritic pain and distress.
- **Laryngeal TB:** To reduce inflammation and improve symptoms.
- **TB Immune Reconstitution Inflammatory Syndrome (IRIS):** In patients with HIV, steroids can help manage the inflammatory response during antiretroviral therapy.

Steroid Dosage

- **Prednisolone:** 1-2 mg/kg/day for 4 weeks, then tapered over the next 4 weeks.
- **Dexamethasone:** 0.6 mg/kg/day for 4 weeks, then tapered over the next 4 weeks.
- **Equivalents:** Any steroid in equipotent doses can be used.

Monitoring and Precautions

- **Monitoring:** Regular monitoring for potential side effects of steroids, including immunosuppression, hyperglycemia, and osteoporosis, is essential.
- **Duration:** The duration of steroid therapy should be tailored to the individual patient's response and the severity of the disease.

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Q: Adverse Effects of First Line Anti-TB Drugs; how will you manage ATT induced liver toxicity

Drug	Main Effects	Rare Effects
Isoniazid	Peripheral neuropathy, skin rash, Hepatitis, Sleepiness & lethargy	Convulsion, Psychosis, Arthralgia, Anemia
Rifampicin	Orange discoloration, pain, nausea, vomiting, Hepatitis, generalized cutaneous reactions, thrombocytopenic purpura	Osteomalacia, Pseudomembranous colitis, Pseudoporphyria, Acute Renal failure, Hemolytic reactions
Pyrazinamide	Arthralgia, hepatitis, Gastrointestinal discomfort	Cutaneous anaemia reactions, Generalised cutaneous reactions, Arthralgia, peripheral neuropathy, hepatitis
Ethambutol	Retrobulbar neuritis	-

Side Effects of Second Line Anti-TB Drugs

Drugs	Side Effects
Injectables: Kanamycin/Capreomycin	Ototoxicity, Nephrotoxicity

Quinolones-Ofloxacin, Levofloxacin, Moxifloxacin	Electrolyte imbalance, Gastro-intestinal-Abdominal pain, nausea, vomiting, CNS-dizziness, convulsions, Phototoxicity, photosensitivity, Tendinopathy, tendinitis, Skin rash, Cardiotoxicity-QT prolongation, Arthralgia
Ethionamide	Gastro-intestinal: epigastric discomfort, anorexia, metallic taste, nausea, vomiting, Excessive salivation, sulfurous belching, Hepatitis, Hypothyroidism and goiter with prolonged administration, Gynaecomastia, menstrual disturbance, impotence, acne, headache, peripheral neuropathy
Cycloserine	CNS dizziness, slurred speech, convulsion, headache, tremor, insomnia, Psychiatric confusion, depression, altered behavior, suicidal tendency, Hypersensitivity reactions

Management of Adverse Drug Reactions (ADR)

1. *Initial Steps:*

- Check the dose of the drugs.
- Exclude other causes of symptoms.
- Estimate the seriousness of the adverse effects.
- Register the adverse effects.
- Stop the drugs if necessary and reintroduce gradually when symptoms disappear.
- Avoid development of drug resistance.

2. *Approach to Side Effects:*

- Classify side effects as major or minor.
- Continue TB treatment with symptomatic treatment for minor adverse effects.
- For major side effects, stop the responsible drug or the entire regimen.
- Urgently refer the patient to a clinician or healthcare facility for further assessment and treatment.
- Manage patients with major adverse reactions in a hospital.

- In drug-resistant TB (DR-TB) patients, involve the DR-TB Committee in the management and modification of the regimen.

ATT Drug-Induced Liver Injury

1. *Identification and Risk Factors:*

- Hepatotoxicity usually occurs within weeks to months, often in the first 3 months of treatment.
- Clinical features include nausea, vomiting, anorexia, abdominal pain, jaundice, unexplained fatigue, new onset hepatomegaly, and bleeding manifestations.
- Risk factors include malnutrition, hypoalbuminemia, HIV, Hepatitis B/C infection, extensive TB disease, age, genetic factors (slow acetylators of INH, HLA-DQB1*0201, gene polymorphisms).

2. *Management:*

- Asymptomatic increases in serum liver transaminases: Continue ATT, monitor for symptoms, and may repeat liver enzymes after a week.
- Drug-induced liver injury (defined as ALT and/or AST >5 times the upper limit of normal or >3 times with symptoms, or serum total bilirubin above 1.5 mg/dl): Stop all hepatotoxic ATT drugs (Rifampicin, Isoniazid, Pyrazinamide) immediately. Provide symptomatic treatment. Check for other causes of hepatitis (viral markers).
- Consider starting alternative drugs if seriously ill (Ethambutol, Streptomycin, Levofloxacin).
- Reintroduce primary drugs once symptoms subside and liver enzymes are <2 times the upper limit of normal (2-6 weeks). Start with full dose of Rifampicin (after LFT is <2ULN). Add drugs every 3 days with regular LFT monitoring. Reintroduce a new drug only if ALT is less than twice the upper limit of normal.
- Refer the patient to an in-patient facility if the patient is sick, drowsy, or has abnormal bleeds.

3. *Duration of Therapy:*

- Do not count the days when the full complement of treatment was not given while deciding the final duration of therapy.

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Q: Paradoxical upgrading reactions

Also known as paradoxical reactions or paradoxical response, are a phenomenon observed in some patients undergoing treatment for tuberculosis (TB).

Paradoxical upgrading reactions represent a complex interplay between the host immune response and TB therapy.

Definition

- **Paradoxical Reaction:** It refers to the enlargement of old lesions or the unexpected appearance of new lesions during apparently adequate antituberculous therapy (ATT).

Timing and Duration

- **Occurrence:** Typically occurs 3–12 weeks after the beginning of therapy, most frequently after 6–7 weeks.
- **Duration:** Lasts for approximately 2 months.
- **Post-Treatment:** May occur post-treatment, as late as two years, especially in cases of lymph node TB.

Characteristics

- **Self-Limiting:** Generally, it is self-limiting and resolves without serious sequelae.
- **Treatment:** Usually regresses without a change in the initial drug regimen.
- **Immune Response:** Active TB can result in depression of immunity. After successful ATT, the focal immune response improves, leading to accumulation of inflammatory exudates at previously invisible microscopic tuberculous foci, appearing as new lesions.
- **Radiographic Progression:** Reversible roentgenographic progression in the initial treatment of TB may be more common than previously expected. Frequent monitoring by chest radiograph is not recommended as it may overdiagnose paradoxical reactions.

Types of Paradoxical Reactions Reported

- Increase in size of mediastinal lymph nodes or areas of pulmonary infiltration in pediatric patients with primary TB.
- Appearance of new lung infiltrates in patients with extrapulmonary TB.

- Development of TB pleural effusion.
- Increase in size of effusion or appearance of effusion on the contralateral side.
- Appearance of new lymph nodes or enlargement of original nodes.
- Increase in size or number of tuberculoma, infarctions, or hydrocephalus on treatment of intracranial TB.

Diagnosis

- **Diagnosis of Exclusion:** It might be difficult to distinguish a paradoxical reaction from true drug resistance, and it is always a diagnosis of exclusion.
- **Absence of Systemic Symptoms:** Absence of systemic symptoms is a useful indicator for paradoxical upgrading reactions. This absence is usually seen in paradoxical reactions (non-HIV settings).

Additional Information

- **Risk Factors:** Risk factors for paradoxical reactions include HIV infection, the severity of TB disease, and rapid response to ATT.
- **Management:** Management primarily involves reassurance and continuation of the current ATT regimen. In severe cases, adjunctive corticosteroids may be considered.
- **Monitoring:** Close monitoring is essential to differentiate between paradoxical reactions and treatment failure or drug resistance.

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Q: Management of a neonate born to a mother with tuberculosis

Managing a neonate born to a mother with tuberculosis (TB) requires a comprehensive approach that includes evaluation for TB, preventive therapy, and considerations for maternal and neonatal care. Here's a detailed management plan based on the provided data:

Evaluation of the Neonate

1. **Clinical Examination:** A thorough clinical examination is essential as symptoms can be subtle.
2. **Chest Radiograph:** Necessary for evaluating pulmonary involvement.

3. **Additional Investigations:** Based on symptoms and examination, other investigations like ultrasound (USG) of the abdomen or gastric aspirate, placental HPE may be indicated.

Factors Affecting Transmission Risk

- **Maternal Disease:** High risk with miliary, meningeal, or pulmonary TB; low with pleural effusion or lymph node TB; unknown with HIV co-infection.
- **Therapy:** Low risk if the mother has completed treatment or has taken at least 2 weeks of treatment.
- **Chemoprophylaxis:** Use of preventive therapy.
- **Contact:** Closeness of contact, isolation, and barrier nursing.

Preventive Therapy for the Neonate

- **Indications:** Recommended for neonates whose mother has any form of active TB detected during pregnancy or after birth, or if the neonate is exposed to any infectious case of TB after birth.
- **Exclusion of Active Disease:** Active disease should be ruled out in neonates.
- **INH Preventive Therapy:** Isoniazid (INH) is given at a dose of 10 mg/kg for a duration of 6 months. Pyridoxine may be given alongside.
- **Chemoprophylaxis in MDR Contacts:** Not recommended due to the uncertain efficacy of second-line drugs in preventing TB and potential toxicity.

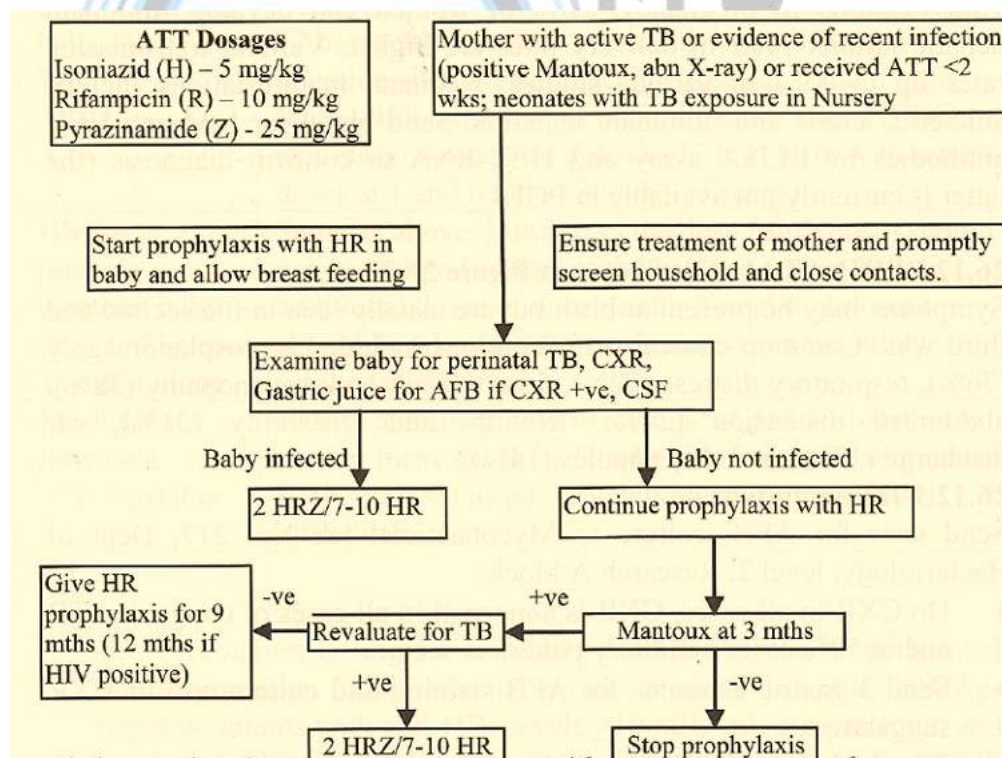
Mother-Infant Interaction

- **Separation:** Not considered mandatory once the mother's therapy is started and if the baby is on isoniazid preventive therapy (IPT). Separation should occur only under specific circumstances, such as the mother being ill enough to require hospitalization, non-adherence to treatment, or infection with a drug-resistant strain.
- **Breastfeeding:** The mother can continue to breastfeed. ATT excreted in breast milk has no therapeutic or adverse effect on the baby. Appropriate cough hygiene should be observed.

Mother with active TB or evidence of recent infection (positive Mantoux, abnormal X-ray) or received ATT <2 weeks; neonates with TB exposure in Nursery:

- Start prophylaxis with HR in the baby and allow breast milk feeding.
- Ensure the treatment of the mother and promptly screen household and close contacts.

- Examine the baby for perinatal TB, CXR, Gastric juice for AFB if CXR is positive, and CSF.
 - If the baby is infected:
 - Administer 2 HRZ/7-10 HR treatment.
 - If the baby is not infected:
 - Continue prophylaxis with HR.
 - Reevaluate for TB if symptoms are present.
 - Perform Mantoux testing at 3 months.
 - If positive, administer 2 HRZ/7-10 HR treatment. (*Note: RNTCP recommends only INH as chemoprophylaxis*)
 - If negative, stop prophylaxis.
- If the mother only shows positive Mantoux results but has a normal X-ray and sputum or becomes asymptomatic once receiving ATT for 2 weeks, provide HR prophylaxis to the baby for 6-9 months (or 12 months if HIV positive).



Vaccination

- **BCG Vaccine:** Decreases the risk of tuberculosis in exposed infants. All children born to mothers with TB should receive BCG at birth, even if they are on isoniazid preventive therapy.

Tuberculin Skin Test (TST)

- **Role in Neonates:** TST has a limited role in neonates and young infants and is hence not recommended.

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Q: Perinatal Tuberculosis

- **Definition:** Congenital TB is acquired during intrauterine life or before complete passage through the birth canal. Perinatal TB encompasses true congenital and neonatal forms of the disease.
- **Infectious Routes:** Transplacental route leading to a primary complex in the liver, and aspiration or ingestion of infected amniotic fluid leading to a primary focus in the lungs or gastrointestinal tract.
- **Diagnostic Criteria:** As described by Cantwell, including lesions in the newborn baby during the first week of life, tuberculous infection of the placenta or maternal genital tract, and exclusion of postnatal transmission.
 - Diagnostic criteria for congenital TB with at least one of the following Lesions in newborn baby during the first week of life
 - A primary hepatic complex or caseating hepatic granulomata
 - Tuberculous infection of placenta or maternal genital tract
 - Exclusion of possibility of postnatal transmission by investigation of contacts, including hospital staff

Limitation to Cantwell criteria

- Presentation may be late; after 2-3 weeks of birth
- Difficulty in demonstration of AFB in neonates if GA is negative
- Inadequate opportunities / facilities for examination of placenta / endometrium
- Percutaneous liver biopsies are also difficult to perform, especially in sick neonates with multiple comorbid factors
- Incomplete evaluation of mother, especially if symptoms of TB are not florid and inadequate evaluation of contacts, including hospital attendants compounds the problem

- Symptoms may manifest at birth but typically appear during the second and third weeks.
- Common clinical manifestations include:
 - Hepatosplenomegaly
 - Respiratory distress (72%)
 - Fever
 - Lymphadenopathy
 - Abdominal distension (24%)
 - Lethargy and irritability
 - Ear discharge (17%)
 - Skin papules (14%)

Investigations:

- Always conduct a CXR. An abnormal CXR indicates congenital TB, with a miliary pattern observed.
- Send three gastric aspirates for AFB staining and culture if the CXR suggests TB.
- Examine the placental tissue for histopathological examination and culture.
- Examine the CSF for AFB stain and culture.
- If available, and if a diagnosis remains uncertain following the aforementioned tests, conduct a lymph node biopsy and bone marrow aspirate.
- Consider HIV testing, especially if TB is confirmed.

Treatment:

- If the mother has active TB or shows signs of recent infection (abnormal X-ray or positive Mantoux results) and if the infant hasn't started ATT (whether preventive or therapeutic), separate the infant and mother.
- If the infant has commenced ATT, breastfeeding is permissible.
- If Multi-drug Resistant (MDR) TB is suspected, or if the mother is noncompliant or has received ATT for less than 2 weeks, isolation of the mother from the baby is advised.
- Express breast milk (EBM) remains permissible even if the mother and infant are separated.

- Administer the BCG vaccine to all infants before hospital discharge. A special INH-resistant BCG isn't necessary.
- If the infant is receiving ATT, administer pyridoxine at a dosage of 5 mg/kg OD.

Treatment Regimen: Similar to pediatric TB.

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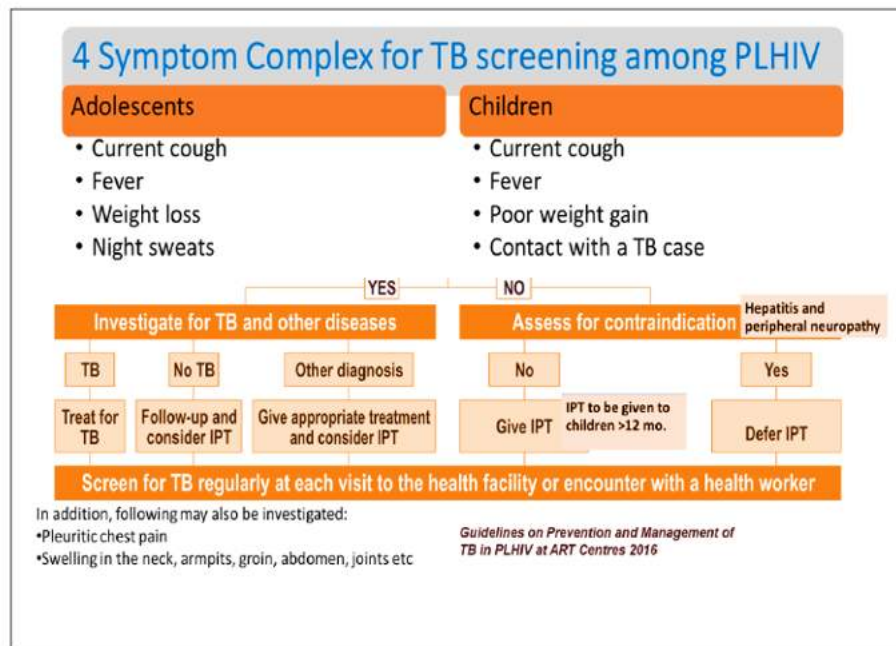
Q: Management of TB with HIV Co-Infection

Epidemiology and Public Health Importance

- HIV epidemic can drive TB epidemic and vice versa.
- About 110,000 people in India are estimated to be HIV-TB co-infected annually.
- High death rates (15-18%) reported among HIV-infected TB cases.
- TB causes about 25% of all deaths among People Living with HIV (PLHIV) in India.

Screening and Testing

- Important to screen children with HIV for TB, and vice versa.
- All patients with TB should be tested for HIV after counseling.
- Rapid HIV tests are available at all Integrated Counseling and Testing Centre (ICTC) centers.



Diagnostic Challenges

- Overlapping symptoms between HIV and TB.
- Difficulty in interpreting chest radiographs (CXR) due to other HIV-related conditions.
- Tuberculin Sensitivity Testing (TST) has low sensitivity in culture-confirmed TB with HIV cases.
- Sputum microscopy has poor sensitivity in detecting TB in PLHIV.

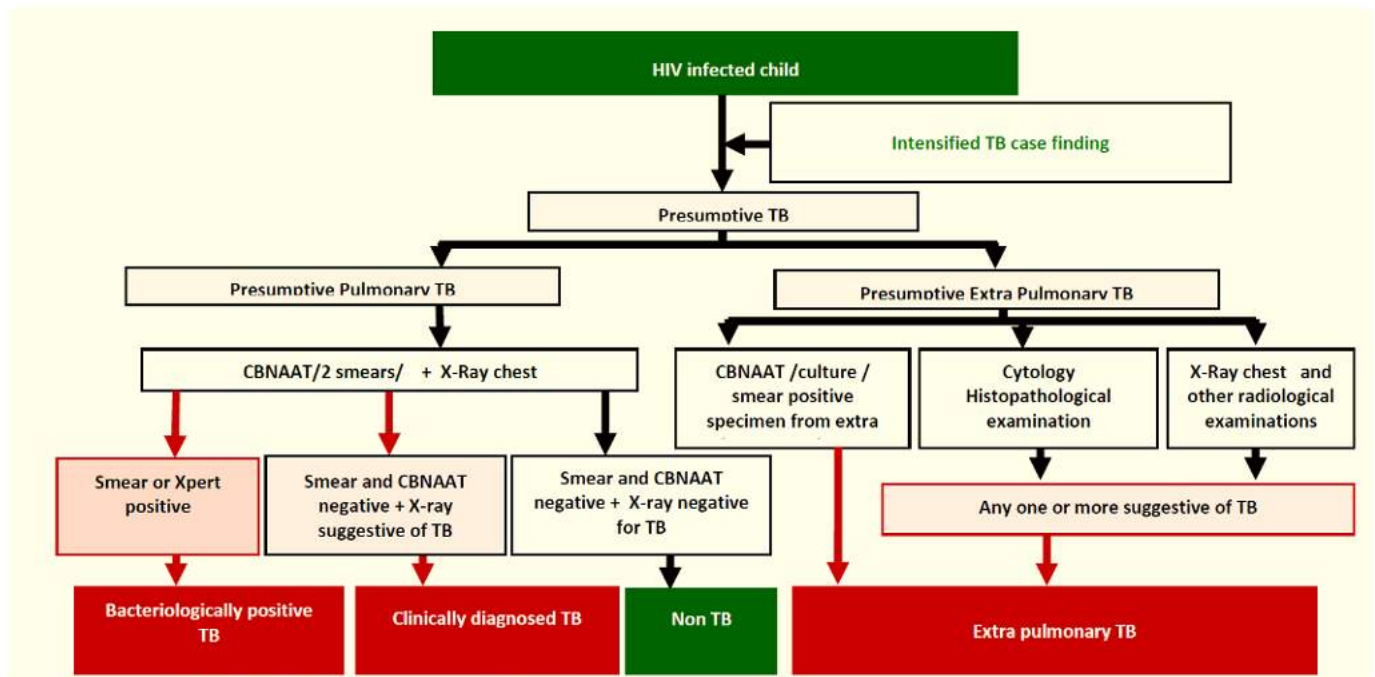
Treatment Challenges

- Treatment is more difficult, and adverse events are more common.
- Deaths during treatment are partly due to TB itself and partly due to HIV-related diseases.
- Increased frequency of adverse drug reactions requires rigorous monitoring.

Diagnostic Algorithm for TB in Children Living with HIV (CLHIV)

- Intensified TB case finding for HIV-infected children.
- Use of CBNAAT (Cartridge Based Nucleic Acid Amplification Test) for diagnosis of TB.
- Bacteriologically positive TB cases are identified through CBNAAT, sputum smear, or chest X-ray.

- Clinically diagnosed TB cases are identified when smear and CBNAAT are negative but X-ray is suggestive of TB.



Strategy for HIV-TB Coordination to Reduce Mortality

- Prevention through Isoniazid preventive therapy (IPT) for all CLHIV.
- Early detection of HIV-TB through 100% coverage of provider-initiated HIV testing and counseling in TB patients.
- Rapid diagnostics for detecting TB and Drug-Resistant TB (DR-TB) in PLHIV.
- Early care and prompt treatment of HIV-TB, including prompt initiation of TB treatment and early initiation of Antiretroviral Therapy (ART).

Strategy for HIV-TB coordination to reduce mortality

Prevention • Isoniazid preventive therapy (IPT) for all CLHIV (on ART+ Pre-ART) • Robust Airborne infection control (AIC) activities • Awareness generation	Early Detection of HIV-TB ✚ 100% coverage of provider-initiated HIV testing and counselling (PITC) in TB patients ✚ PITC in presumptive TB cases ✚ Rapid diagnostics for detecting TB and DR-TB in PLHIV
Early Care & Prompt Treatment of HIV-TB ❖ Promotion of 'Single Window Delivery Services' i.e. ATT from ART centre along with ART drugs ❖ Prompt initiation of TB treatment ❖ Early initiation of ART (within first 8 wks) & monitoring of timeliness of ART initiation through expanded ART reporting formats ❖ Strengthened linkage of HIV-TB patients to ART centres through travel support by RNTCP ❖ ART for HIV-TB cases irrespective of CD4 count	✚ Intensive Case Finding (ICF) activities at all HIV settings (ICTCs, ART centres, Link ART Centre (LACs), and Targeted Intervention (TI settings): all ICTC clients should be screened for presence of TB and vice-versa

TB Treatment Recommendations

- Treatment regimen is the same as in HIV non-infected children.
- 6 months duration of therapy is optimal, with intensive and continuation phases.
- Pyridoxine supplementation should be given till the time Isoniazid is prescribed in Drug-Susceptible TB (DSTB) co-infected children.

ART among CLHIV with TB Co-Infection

- Management of HIV-TB co-infection in infants, children, and adolescents in relation to age groups and body weight.
- Nevirapine is no longer part of the first-line ART.

Monitoring During Treatment

- HIV-infected children on Anti-Tuberculosis Treatment (ATT): Liver Function Tests (LFTs) at baseline, day 15, month 1, and month 3.
- Guidelines for ATT induced liver disease remain the same.

Initiating ART in Patients with DR-TB

- Use of ART in HIV-infected patients with TB improves survival.
- Second-line anti-TB drugs should be initiated first, followed by ART as soon as second-line anti-TB drugs are tolerated.

- Co-trimoxazole can be provided to all patients with HIV to reduce mortality by preventing opportunistic infections.

TB IRIS (Immune Reconstitution Inflammatory Syndrome)

- After initiation of ART, HIV-1 replication is greatly reduced, resulting in increased CD4 cell function & improved immunity.
- IRIS can manifest in several ways, including new/enlarging lymph nodes, worsening radiological features, and new/worsening CNS tuberculosis.
- IRIS is also a diagnosis of exclusion.

IRIS : Onset of TB-associated IRIS should be within 3 months of starting ART with at least 1 major criterion & 2 minor criteria:

Major criteria

- New/enlarging lymph nodes or other focal tissue enlargement
- New/worsening radiological features
- New/worsening CNS tuberculosis
- New/worsening serositis

Minor criteria

- New/worsening constitutional symptoms such as fever
- New/worsening respiratory symptoms such as cough
- New/worsening abdominal pain

BCG in Children with HIV

- WHO recommendations on BCG in children with HIV.
- BCG is useful for children born in TB-endemic countries but is associated with disseminated BCG disease in vaccinated HIV-infected infants.

Isoniazid Preventive Therapy (IPT) for Children Living with HIV

- IPT is recommended for prevention of incident TB among HIV-infected individuals/children.
- Children living with HIV who do not have poor weight gain, fever, or current cough are unlikely to have active TB.
- Providing IPT to CLHIV does not increase the risk of developing INH resistance TB later.

Algorithm for Use of IPT for Children Living with HIV

- Screening for TB with symptoms such as poor weight gain, fever, and current cough.
- Investigation for TB and other diseases if symptoms are present.
- IPT is given in a dose of 10 mg/kg orally every day for 6 months duration.

(the discussion on tuberculosis has been a little exhaustive but that is a very hot topic for exam)

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Q: Clinical, Radiological features and confirmatory laboratory tests for congenital syphilis

Congenital syphilis is a serious, potentially life-threatening infection in newborns, caused by the bacterium *Treponema pallidum*, which is transmitted from an infected mother to her baby during pregnancy.

Clinical Features

1. **Early Signs (Present at Birth or within First Few Weeks):**
 - Snuffles (nasal discharge).
 - Skin lesions, including vesicular or bullous rash, especially on palms and soles.
 - Hepatosplenomegaly (enlarged liver and spleen).
 - Jaundice.
 - Lymphadenopathy (swollen lymph nodes).
 - Anemia.
 - Thrombocytopenia (low platelet count).
2. **Late Signs (May Present Weeks to Months After Birth):**
 - Failure to thrive.
 - Hutchinson's teeth (notched, widely spaced teeth).
 - Mulberry molars (abnormal molar teeth).
 - Saddle nose (deformity of the nose).

- Saber shins (curvature of the tibia).
- Interstitial keratitis (inflammation of the cornea leading to vision problems).
- Sensorineural hearing loss.
- Neurological symptoms (such as seizures or developmental delays).

Radiological Features

- **Bone Changes:** Radiographs may show characteristic changes, especially in long bones, such as metaphyseal dystrophy, periostitis, and osteochondritis.
- **Other Findings:** May include dactylitis (inflammation of fingers and toes) and other skeletal abnormalities.

Confirmatory Laboratory Tests

1. Serologic Tests:

- Non-treponemal tests (e.g., Rapid Plasma Reagin [RPR], Venereal Disease Research Laboratory [VDRL] test): Used for screening. Titers are usually high in congenital syphilis.
- Treponemal tests (e.g., Fluorescent treponemal antibody absorption [FTA-ABS], T. pallidum particle agglutination [TP-PA]): Used for confirmation. These tests remain positive for life, even after treatment.

2. Direct Detection:

- Darkfield microscopy: Can be used to visualize T. pallidum in lesion exudates or tissue.
- Polymerase Chain Reaction (PCR): Highly sensitive and specific for detecting T. pallidum DNA.

3. Other Tests:

- Complete blood count (CBC): May show anemia and thrombocytopenia.
- Liver function tests: May show evidence of hepatitis.

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Q: Management of congenital syphilis

1. Antibiotic Therapy:

- **Penicillin G:** The mainstay of treatment for congenital syphilis. The specific type (aqueous crystalline penicillin G, procaine penicillin, or benzathine penicillin G) and duration of therapy depend on the clinical manifestations and stage of the disease.
- **For Asymptomatic Infants (or those with only evidence of congenital syphilis on physical examination):** A single intramuscular dose of benzathine penicillin G is often recommended.
- **For Infants with Clinical Signs of Congenital Syphilis or Those Born to Mothers with Untreated or Inadequately Treated Syphilis:** Aqueous crystalline penicillin G, administered intravenously, is typically used for 10-14 days.
- **For Infants with Neurosyphilis or Uncertain Treatment Response:** Treatment may involve a longer course of aqueous crystalline penicillin G.

Clinical Scenario	Antibiotic	Formulation	Dosage	Duration
Asymptomatic Infants (or those with only evidence of congenital syphilis on physical examination)	Benzathine Penicillin G	Intramuscular	50,000 units/kg (maximum: single dose of 2.4 million units)	Single Dose
Infants with Clinical Signs of Congenital Syphilis or Those Born to Mothers with Untreated or Inadequately Treated Syphilis	Aqueous Crystalline Penicillin G	Intravenous	≤7 days of age: 50,000 units/kg/dose every 12 hours ≥8 days of age: 50,000 units/kg/dose every 8 hours	10-14 Days
Infants with Neurosyphilis or Uncertain Treatment Response	Aqueous Crystalline Penicillin G	Intravenous	≤7 days of age: 50,000 units/kg/dose every 12 hours ≥8 days of age: 50,000	10-14 Days (may require longer treatment)

			units/kg/dose every 8 hours	
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2. Evaluation and Monitoring:

- **Thorough Physical Examination:** To assess the extent of the disease.
- **Serologic Testing:** Follow-up serologic testing is essential to ensure an appropriate response to therapy. Non-treponemal titers should decrease following treatment.
- **Neurological Evaluation:** Including cerebrospinal fluid (CSF) analysis, especially in infants with abnormal neurologic findings or those born to mothers with untreated syphilis.
- **Ophthalmologic Examination:** To rule out ocular involvement.
- **Audiologic Examination:** To assess for hearing loss.

3. Follow-Up:

- **Regular Follow-Up Visits:** To monitor clinical, serologic, and developmental progress.
- **Repeat Serologic Testing:** At 3, 6, and 12 months to ensure that titers are decreasing.
- **Repeat CSF Analysis:** If initial CSF analysis was abnormal, repeat testing every 6 months until normal.

4. Management of the Mother:

- **Treatment of the Mother:** If the mother was not adequately treated during pregnancy, she should receive treatment for syphilis.
- **Partner Notification and Treatment:** Sexual partners of the mother should also be notified, tested, and treated as appropriate.

5. Prevention:

- **Prenatal Screening:** All pregnant women should be screened for syphilis early in pregnancy and again at delivery if at high risk. Mandatory under Govt. of India MOH rules.
- **Education:** Educating parents about the importance of follow-up and the potential long-term complications of congenital syphilis.

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Q: Diagnosis and management of Leptospirosis

- **Incubation Period:** Ranges from 2 to 30 days.
- **Clinical Manifestations:**
 - Can range from asymptomatic to severe disease with multiorgan dysfunction.
 - Abrupt onset, potentially following a monophasic or biphasic course.
 - Initial phase (septicemic): Lasts 2-7 days, with leptospires isolatable from blood, CSF, and other tissues.
 - Possible brief asymptomatic period.
 - Second phase (immune or leptospiruric): Characterized by circulating IgM antibody, disappearance of organisms from blood and CSF, and symptoms related to tissue localization of leptospires. This phase can last several weeks.
 - Leptospires can persist in the kidney, urine, and aqueous humor despite circulating antibodies.
- **Symptomatic Infection:** Can be anicteric or icteric.

Treatment of Leptospirosis

- **Antibiotic Therapy:**
 - In vitro susceptibility to penicillin and tetracyclines.
 - In vivo effectiveness unclear due to high spontaneous recovery rates.
 - Early treatment (before the 7th day) may shorten the clinical course and decrease severity.
 - Recommended antibiotics: Penicillin G, cefotaxime, ceftriaxone, or doxycycline (for children ≥ 8 years).
 - Doxycycline can be used safely in children >2 years for short courses.
 - Parenteral penicillin G: 6-8 million U/m²/day divided every 4 hours IV for 7 days.
 - Doxycycline alternative: 2 mg/kg/day divided in 2 doses, maximum 100 mg twice daily.

- Alternatives for penicillin-allergic patients: Cefotaxime, ceftriaxone, and azithromycin.
- Mild illness: Oral doxycycline, amoxicillin, and ampicillin.
- Severe illness: Supportive care focusing on cardiopulmonary status, renal function, coagulopathy, and fluid and electrolyte balance.

Prevention and Prophylaxis

- **Prevention:**
 - Rodent control measures.
 - Avoidance of contaminated water and soil.
 - Immunization of livestock and domestic dogs.
 - Protective clothing for persons at risk for occupational exposure.
 - Hospital settings: Standard precautions and contact precautions for potential exposures to infected urine.
- **Prophylaxis:**
 - Successful prevention in American soldiers stationed in the tropics with doxycycline (200 mg PO once a week).
 - Potential effectiveness for travelers to highly endemic areas for short periods.
 - Lack of specific pediatric data to support any prophylaxis regimen.

Additional Considerations

- **Diagnostic Challenges:** Due to the wide range of clinical manifestations and the potential for asymptomatic infections.
- **Geographical Variability:** The diversity of *Leptospira* serovars and their variable geographic distribution complicates vaccine development and prophylaxis strategies.
- **Occupational Risks:** Particular attention should be given to individuals with occupational exposure to potentially contaminated environments.

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Q: Clinical Presentation and Diagnosis of Mycoplasma Pneumonia Infection

Clinical Presentation

- **Onset:** Usually gradual, characterized by headache, malaise, fever, and sore throat, followed by lower respiratory symptoms including hoarseness and nonproductive cough. In some cases, the onset may be abrupt.
- **Symptoms:** Cough, the clinical hallmark, usually worsens during the first week of illness and can last up to 4 weeks, sometimes accompanied by wheezing.
- **Physical Examination:** Chest examination may reveal minimal findings, even in patients with severe cough. Auscultative or percussive findings may be absent or minimal, with dry rales occasionally present.
- **Radiographic Findings:** Variable and nonspecific. Pneumonia is usually described as interstitial or bronchopneumonic, with common involvement in the lower lobes. Bilateral diffuse infiltrates, lobar pneumonia, or hilar lymphadenopathy can occur in up to 30% of patients. Large pleural effusions, necrotizing pneumonia, and bronchiolitis obliterans are rare complications.
- **Extrapulmonary Manifestations:** CNS disease, dermatologic disease, hematologic abnormalities, and involvement of other organs like the heart, gastrointestinal tract, and joints can occur.

Diagnosis

- **Diagnostic Challenges:** No definitive clinical or radiographic findings for *M. pneumoniae* infection. Gradual onset and cough as prominent findings in school-age children and young adults suggest *M. pneumoniae* infection.
- **Laboratory Tests:**
 - PCR from respiratory samples and serology (acute and convalescent) are the best methods for diagnosis.
 - Cultures on special media (SP4 agar media) of the throat or sputum might demonstrate *M. pneumoniae* colonies, but growth generally requires incubation for 2-3 weeks, making it impractical.
 - Serologic tests (immunofluorescence tests, enzyme-linked immune assays [EIAs], or complement fixation) to detect serum immunoglobulin (Ig) M and IgG antibodies against *M. pneumoniae* are commercially available.

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Q: Diagnosis and Treatment of Scrub Typhus

Clinical Presentation and Laboratory Findings

- Scrub typhus can range from mild to severe in children, affecting almost every organ system.
- Common presentation includes fever for 9-11 days before seeking medical care.
- Regional or generalized lymphadenopathy is reported in 23-93% of patients.
- Hepatomegaly in about two-thirds, and splenomegaly in about one-third of children.
- Gastrointestinal symptoms such as abdominal pain, vomiting, and diarrhea occur in up to 40% of children.
- A single painless eschar with an erythematous rim at the site of the chigger bite is seen in 7-68% of cases.
- A maculopapular rash is present in less than half of the cases; both eschar and rash can be absent.
- Hemophagocytic lymphohistiocytosis has been described.
- Leukocyte and platelet counts are most commonly within normal ranges, although thrombocytopenia occurs.

Differential Diagnosis

- Differential diagnosis includes fever of unknown origin, enteric fever, typhoid fever, dengue hemorrhagic fever, other rickettsioses, tularemia, anthrax, dengue, leptospirosis, malaria, and infectious mononucleosis.

Treatment and Supportive Care

- The recommended treatment regimen for scrub typhus is doxycycline (4 mg/kg/day PO or IV divided every 12 hr; maximum: 200 mg/day).
- Treatment should be continued for a minimum of 5 days and until the patient has been afebrile for at least 3 days to avoid relapse.
- A single dose of oral doxycycline was reported effective for all 38 children treated with this regimen in a large series of children with scrub typhus from Thailand.
- Most children respond rapidly to doxycycline or chloramphenicol within 1-2 days (range: 1-5 days).

- Strains of *O. tsutsugamushi* with modestly higher doxycycline minimal inhibitory concentrations are reported in some regions of Thailand.
- Clinical trials showed that azithromycin could be as effective, and that rifampicin is superior to doxycycline in such cases and could have a role as an alternative therapy, especially for pregnant women.
- The use of ciprofloxacin in pregnant women resulted in an adverse outcome in 5 of 5 pregnancies among Indian women.
- Intensive care may be required for hemodynamic management of severely affected patients.

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Q: Laboratory findings and diagnosis of rickettsial infection

Laboratory Findings in Rickettsial Infections:

1. Complete Blood Count (CBC):

- Mild leukopenia or leukocytosis
- Thrombocytopenia is common

2. Biochemical Tests:

- Elevated liver enzymes (AST, ALT)
- Hyponatremia
- Elevated creatinine levels

3. Serology:

- Weil-Felix test: Historically used, but has low sensitivity and specificity
- Indirect immunofluorescence assay (IFA): Gold standard for diagnosis
- Enzyme-linked immunosorbent assay (ELISA): Also used for antibody detection

4. Molecular Tests:

- Polymerase chain reaction (PCR): Useful for early diagnosis, can detect rickettsial DNA in blood or tissue samples

5. Skin Biopsy (if rash is present):

- Immunohistochemical staining can detect rickettsial organisms in tissue

6. **Other Findings:**

- Cerebrospinal fluid (CSF) analysis may show mild pleocytosis
- Elevated C-reactive protein (CRP) and erythrocyte sedimentation rate (ESR)

Diagnostic Approach:

1. **Clinical Presentation:**

- Consider rickettsial infection in patients with fever, rash, and history of tick or flea exposure

2. **Initial Laboratory Tests:**

- CBC, liver function tests, electrolytes, renal function tests

3. **Serological Testing:**

- IFA or ELISA to detect antibodies against Rickettsia species
- Acute and convalescent sera should be tested to demonstrate seroconversion or a significant rise in antibody titers

4. **Molecular Testing:**

- PCR can be performed on blood, skin biopsy, or other tissue samples

5. **Skin Biopsy:**

- In cases with rash, a skin biopsy can be helpful for direct detection of the organism

6. **Imaging Studies:**

- May be indicated based on clinical presentation (e.g., chest X-ray for respiratory symptoms)

7. **Differential Diagnosis:**

- Consider other causes of fever and rash, including viral exanthems, bacterial infections, and other vector-borne diseases

8. **Consultation and Reporting:**

- Consult infectious disease specialist for complex cases
- Report cases to public health authorities as required

9. **Follow-up:**

- Monitor response to treatment and adjust as necessary
- Repeat serological testing in convalescent phase to confirm diagnosis

Key Points:

- Early recognition and treatment are crucial to prevent complications.
- Diagnosis is often based on clinical presentation and epidemiological factors, supported by laboratory findings.
- Serology is the mainstay of diagnosis but may not be positive in the early stages of the disease.
- PCR can provide early diagnosis but is not widely available.
- A high index of suspicion is necessary, especially in endemic areas or during the tick season

Q: Treatment of Rickettsial Infections:**1. Antibiotic Therapy:**

- **Doxycycline:**
 - First-line treatment for most rickettsial infections.
 - Dosage for adults: 100 mg orally or intravenously every 12 hours.
 - Dosage for children: 2.2 mg/kg body weight (up to 100 mg) orally or intravenously every 12 hours.
 - Duration: Typically 7-14 days, depending on the severity and clinical response.
- **Chloramphenicol:**
 - Alternative for patients who cannot tolerate doxycycline.
 - Dosage for adults: 500 mg orally or intravenously every 6 hours.
 - Dosage for children: 12.5 mg/kg body weight orally or intravenously every 6 hours.
 - Monitor for bone marrow suppression.

2. Supportive Care:

- **Fluid and Electrolyte Management:**

- Correct dehydration and electrolyte imbalances.
- **Fever Control:**
 - Antipyretics for fever and discomfort.
- **Nutritional Support:**
 - Ensure adequate nutrition and hydration.

3. Management of Complications:

- **Respiratory Support:**
 - Oxygen therapy or mechanical ventilation for severe respiratory involvement.
- **Renal Support:**
 - Dialysis for acute renal failure.
- **Cardiovascular Support:**
 - Inotropic support for myocarditis or shock.

4. Monitoring and Follow-up:

- **Clinical Monitoring:**
 - Regular assessment of vital signs, organ function, and response to treatment.
- **Laboratory Monitoring:**
 - Repeat CBC, liver and renal function tests, and other relevant investigations.
- **Follow-up Visits:**
 - After completion of therapy to ensure resolution of symptoms and to monitor for any sequelae.

5. Prevention and Control:

- **Vector Control:**
 - Measures to reduce exposure to ticks, fleas, and mites.
- **Personal Protective Measures:**
 - Use of insect repellents, protective clothing, and avoidance of known endemic areas.

- **Public Health Measures:**

- Surveillance and reporting of cases to public health authorities.

6. Special Considerations:

- **Pregnant Women:**

- Doxycycline is generally avoided; consult with an infectious disease specialist.

- **Severe or Life-threatening Infections:**

- Consider hospitalization and intravenous administration of antibiotics.

Key Points:

- Early initiation of appropriate antibiotic therapy is crucial for favorable outcomes.
- Doxycycline is the antibiotic of choice for most rickettsial infections.
- Supportive care is essential to manage symptoms and complications.
- Monitor for adverse effects of antibiotics and adjust treatment as necessary.
- Prevention strategies are important to reduce the risk of infection.

Q: When to Suspect Rickettsial Infection in Children:

- **Epidemiological Factors:**

- Exposure to endemic areas or recent travel history.
- Contact with vectors such as ticks, fleas, or mites.

- **Clinical Presentation:**

- Fever of unknown origin.
- Rash, which may be maculopapular, petechial, or purpuric.
- History of tick or insect bite, sometimes with an eschar.
- Headache, myalgia, and arthralgia.
- Gastrointestinal symptoms like nausea, vomiting, or diarrhea.

- Respiratory symptoms in some cases.
- Neurological symptoms such as confusion or meningismus.
- **Laboratory Findings:**
 - Thrombocytopenia or leukopenia.
 - Elevated liver enzymes.
 - Hyponatremia.
- **Non-Response to Usual Antibiotics:**
 - Lack of improvement with common antibiotics used for bacterial infections.

Different Forms of Rickettsial Infections in Children:

Disease	Causative Agent	Vector	Clinical Features	Geographic Distribution
Rocky Mountain Spotted Fever	<i>Rickettsia rickettsii</i>	Ticks	Fever, rash (starts on wrists and ankles), headache, myalgia	Americas, especially the United States
Mediterranean Spotted Fever	<i>Rickettsia conorii</i>	Ticks	Fever, rash, eschar, headache	Mediterranean region, Africa, India
Scrub Typhus	<i>Orientia tsutsugamushi</i>	Chiggers	Fever, eschar, rash, lymphadenopathy, hepatosplenomegaly	Asia-Pacific region
Epidemic Typhus	<i>Rickettsia prowazekii</i>	Lice	Fever, rash (starts on trunk), headache, myalgia	Africa, South America, high altitude areas
Murine Typhus	<i>Rickettsia typhi</i>	Fleas	Fever, rash, headache, myalgia	Worldwide, especially in port cities
Anaplasmosis	<i>Anaplasma phagocytophilum</i>	Ticks	Fever, headache, myalgia, leukopenia, thrombocytopenia	North America, Europe

Ehrlichiosis	<i>Ehrlichia species</i>	Ticks	Fever, headache, myalgia, leukopenia, thrombocytopenia	United States, Europe
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Q: Clinical Features and Stages of Subacute Sclerosing Panencephalitis (SSPE)

Clinical Features:

- **Onset and Progression:**
 - SSPE begins insidiously 7-13 years after primary measles infection.
 - Initial symptoms include subtle changes in behavior or school performance.
- **Stage I:**
 - Normal EEG patterns.
 - Early signs may be subtle and non-specific.
- **Stage II (Myoclonic Phase):**
 - Characterized by myoclonic jerks.
 - EEG shows suppression-burst episodes, characteristic but not pathognomonic for SSPE.
- **Stage III:**
 - Further deterioration into dementia, stupor, and then coma.
- **Stage IV:**
 - Loss of critical centers that support breathing, heart rate, and blood pressure.
 - Death usually ensues.
- **Progression:**
 - Can follow courses characterized as acute, subacute, or chronic progressive.

Stages of SSPE:

- **Initial Stage:**
 - Subtle behavioral changes and decline in school performance.

- **Myoclonic Phase:**
 - Myoclonic jerks become prominent.
 - EEG changes with suppression-burst episodes.
- **Advanced Stages:**
 - Progression to dementia, stupor, and coma.
 - Final stage involves loss of autonomic control mechanisms leading to death.
- **Variability in Progression:**
 - The progression through these stages can vary from acute to chronic.

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Q: Diagnosis and Treatment of Subacute Sclerosing Panencephalitis (SSPE)

Pathogenesis of SSPE:

- **Defective Measles Virus and Immune Interaction:**
 - The virus isolated from brain tissue of patients with SSPE is missing the matrix or M protein, responsible for assembly, orientation, and alignment of the virus during viral replication.
 - Immature virus may reside and possibly propagate within neuronal cells for long periods.
 - Most patients with SSPE were exposed at a young age, suggesting that immune immaturity is involved in pathogenesis.

Epidemiology:

- **Incidence:**
 - Rare disease, generally follows the prevalence of measles in a population.
- **Demographics:**
 - Primarily a disease of children and adolescents.
 - Males are affected twice as often as females.
 - Higher prevalence among children of Hispanic origin.

Diagnosis of SSPE:

- **Clinical Course Documentation:**

- Compatible clinical course is essential for diagnosis.
- SSPE typically begins insidiously 7-13 years after primary measles infection.

- **Supporting Findings:**

- Measles antibody detected in cerebrospinal fluid (CSF).
- Characteristic electroencephalographic (EEG) findings.
- Typical histologic findings in and/or isolation of virus or viral antigen from brain tissue obtained by biopsy or postmortem examination.

- **CSF Analysis:**

- Normal cells but elevated IgG and IgM antibody titers in dilutions >1:8.

- **EEG Patterns:**

- Normal in stage I, but in the myoclonic phase, suppression-burst episodes are seen.

- **Brain Biopsy:**

- No longer routinely indicated for diagnosis of SSPE.

Management of SSPE:

- **Primarily Supportive Care:**

- Similar to care provided to patients with other neurodegenerative diseases.

- **Clinical Trials:**

- Isoprinosine with or without interferon suggested significant benefit (30–34% remission rate) compared with patients without treatment (5–10% with spontaneous remissions).

- **Control of Myoclonic Jerks:**

- Carbamazepine is of significant benefit in the control of myoclonic jerks in the early stages of the illness.

- **Prognosis:**

- Virtually all patients eventually succumb to SSPE.

- Most die within 1-3 years of onset from infection or loss of autonomic control mechanisms.
- **Prevention:**
 - Depends on prevention of primary measles infection through vaccination.

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Q: Diagnosis of Congenital Rubella Syndrome (CRS)

- **Clinical Manifestations:**
 - CRS symptoms include cataracts, heart defects, hearing impairment.
 - CRS is chronic, with virus persistence in fetal tissue post-delivery.
- **Diagnostic Testing:**
 - PCR testing of amniotic fluid for prenatal diagnosis.
 - Newborn urine, cord blood, or placenta testing for postnatal diagnosis.
- **Differential Diagnoses:**
 - Differentiate from measles, adenoviruses, parvovirus B19, Epstein-Barr virus, enteroviruses, roseola, Mycoplasma pneumoniae.

Prevention of Congenital Rubella Syndrome (CRS)

- **Isolation:**
 - Isolate postnatal infection patients from susceptible individuals for 7 days post-rash.
 - Children with CRS may excrete virus for up to 1 year; maintain contact precautions.
- **Exposure Management:**
 - Susceptible pregnant women exposed to rubella should have blood tests for rubella IgG-specific antibody.
 - Counseling on risks and benefits of pregnancy termination if susceptible.
- **Immunoglobulin Use:**
 - Routine use for susceptible pregnant women not recommended.

- Consider only if pregnancy termination not an option; may reduce risk for clinically apparent infection but not guarantee fetal infection prevention.
- **Vaccination:**
 - Rubella vaccine, usually with measles and mumps (MMR) or varicella (MMRV).
 - Not for severely immunocompromised patients or during pregnancy.
 - Counseling on theoretical risks to fetus if pregnancy occurs within 28 days of immunization.

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Q: Acute Flaccid Paralysis (AFP) Definition, Differential Diagnosis, and Role in Polio Eradication

Definition of AFP:

- **Acute Flaccid Paralysis (AFP):** A clinical syndrome characterized by rapid onset of weakness, often progressing to maximum severity within several days. It primarily affects the motor neurons and results in flaccid (limp) muscle tone.

Differential Diagnosis of AFP:

- **Poliovirus Infection:** The classic cause of AFP. Characterized by asymmetric limb weakness, fever, and absence of sensory loss.
- **Non-polio Enteroviruses:** Can cause similar symptoms to poliovirus.
- **Guillain-Barré Syndrome (GBS):** Rapidly progressive limb weakness, areflexia, and ascending paralysis. Often follows a respiratory or gastrointestinal infection.
- **Transverse Myelitis:** Inflammation of the spinal cord leading to weakness, sensory alterations, and autonomic dysfunction.
- **Traumatic Neuritis:** Nerve damage due to injury, presenting with localized weakness and pain.
- **Tick Paralysis:** Caused by neurotoxins from tick bites, leading to ascending paralysis.
- **Hypokalemic Periodic Paralysis:** Episodes of muscle weakness associated with low potassium levels.

- **Botulism:** Caused by toxins from *Clostridium botulinum*, leading to descending paralysis.
- **Spinal Muscular Atrophy:** Genetic disorder affecting motor neurons, leading to muscle weakness and atrophy.
- **Myasthenia Gravis:** Autoimmune disorder causing muscle weakness due to impaired neuromuscular transmission.
- **Toxic Neuropathies:** Exposure to toxins or drugs causing nerve damage and weakness.
- **Rabies:** Progressive paralysis following a rabid animal bite.

Role in Polio Eradication:

- **Surveillance Tool:** AFP surveillance is a key strategy in polio eradication efforts. It involves the detection and investigation of all cases of AFP in children under 15 years of age.
- **Case Investigation:** Each AFP case is thoroughly investigated to determine if poliovirus is the cause.
- **Laboratory Testing:** Stool samples from AFP cases are tested for poliovirus. The absence of poliovirus helps in certifying regions as polio-free.
- **Outbreak Response:** Detection of poliovirus in AFP cases triggers immediate outbreak response activities, including mass immunization campaigns.
- **Monitoring Progress:** AFP surveillance serves as an indicator of the effectiveness of polio eradication initiatives. A decline in AFP cases associated with poliovirus indicates progress towards eradication.
- **Global Health Indicator:** AFP surveillance is a global health indicator used by organizations like the World Health Organization (WHO) to monitor and certify the eradication of polio.

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Q: AFP surveillance

- ✚ Acute Flaccid Paralysis (AFP) surveillance is a critical component of the global polio eradication initiative
- ✚ It involves the systematic search for and reporting of cases of AFP in children under 15 years of age, as well as the investigation and classification of these cases to determine if they are caused by the poliovirus

- ✦ AFP surveillance is a cornerstone of the global effort to eradicate polio and is crucial for maintaining a polio-free status after eradication has been achieved
- ✦ It also provides a platform for surveillance of other vaccine-preventable diseases and strengthens overall public health surveillance systems.
- ✦ Acute Flaccid Paralysis (AFP) surveillance, primarily established for polio eradication, also serves as a tool for identifying and monitoring various other causes of AFP
- ✦ This broader approach is crucial for differential diagnosis and public health management

Objectives:

- **Early Detection:** To detect poliovirus transmission in a timely manner.
- **Certification of Polio Eradication:** To provide evidence for the certification of polio eradication.
- **Monitoring of Non-Polio AFP:** To monitor the incidence of non-polio causes of AFP.

Key Components:

- **Case Detection:** Identifying children under 15 years with AFP.
- **Reporting:** Immediate notification of AFP cases to health authorities.
- **Case Investigation:** Detailed investigation of each AFP case within 48 hours of notification.
- **Specimen Collection:** Collection of two stool samples within 14 days of onset of paralysis, and preferably within 24 hours of case notification.
- **Laboratory Analysis:** Testing of stool samples for poliovirus at a WHO-accredited laboratory.
- **Classification:** Classification of cases after 60 days of follow-up to determine if they are polio or non-polio AFP.
- **Data Management and Analysis:** Recording and analysis of data to monitor AFP rates and stool sample adequacy.

Performance Indicators:

- **Non-Polio AFP Rate:** At least 1 case of non-polio AFP per 100,000 children aged <15 years annually.
- **Stool Adequacy:** At least 80% of AFP cases should have two adequate stool specimens collected 24-48 hours apart and within 14 days of paralysis onset.

Challenges:

- **Underreporting:** Ensuring all cases of AFP are reported.
- **Timeliness:** Timely investigation and specimen collection.
- **Differential Diagnosis:** Differentiating polio from other causes of AFP.
- **Resource Allocation:** Ensuring adequate resources for surveillance activities.

Importance:

- **Eradication of Polio:** Essential for the global initiative to eradicate polio.
- **Public Health:** Helps in understanding the epidemiology of other causes of AFP.
- **Health System Strengthening:** Contributes to strengthening overall disease surveillance systems.

List of extended causes of AFP that are important to include in surveillance:

1. **Guillain-Barré Syndrome (GBS):** An autoimmune disorder causing muscle weakness and paralysis.
2. **Transverse Myelitis:** Inflammation of the spinal cord leading to neurological deficits.
3. **Enteroviral Infections Other Than Polio:** Such as enterovirus 71, associated with hand, foot, and mouth disease.
4. **Traumatic Neuritis:** Nerve damage due to injury.
5. **Hypokalemic Paralysis:** Caused by a sudden drop in potassium levels.
6. **Tick Paralysis:** Resulting from neurotoxins released by tick bites.
7. **Rabies (Post-rabies Vaccine Neuralgia):** Rarely, paralysis can occur after rabies or its vaccine.
8. **Peripheral Neuropathy:** Including hereditary and acquired neuropathies.
9. **Myasthenia Gravis:** An autoimmune disorder affecting neuromuscular junctions.
10. **Spinal Muscular Atrophy:** A genetic disorder affecting motor neurons in the spinal cord.
11. **Botulism:** Caused by toxins from *Clostridium botulinum*.
12. **Acute Toxic Neuropathies:** Such as from heavy metal poisoning.

13. **Infectious Myelitis**: Including viral, bacterial, parasitic, and fungal infections.
14. **Post-Infectious Encephalomyelitis**: Following viral or bacterial infections.
15. **Neurological Complications of Systemic Illnesses**: Such as lupus, sarcoidosis.
16. **Vaccine-Associated Paralytic Poliomyelitis (VAPP)**: Rarely associated with the oral polio vaccine.
17. **Congenital Myopathies and Muscular Dystrophies**: Presenting early in life with muscle weakness.
18. **Spinal Cord Compression**: Due to tumors, abscesses, or herniated discs.
19. **Metabolic Disorders**: Such as hypoglycemia or hyperglycemia causing muscle weakness.
20. **Critical Illness Polyneuropathy and Myopathy**: In patients with severe systemic illness.

Importance in Surveillance:

- **Differential Diagnosis**: Helps in distinguishing polio from other causes of AFP.
- **Public Health Management**: Assists in identifying and managing outbreaks of non-polio AFP.
- **Resource Allocation**: Guides healthcare resources to appropriate areas.
- **Epidemiological Tracking**: Monitors trends and patterns in AFP causes.

Q: Discuss in tabular form the differential diagnosis and management of acute acid paralysis in a 2-year-old child

Differential Diagnosis	Clinical Features	Management
Poliovirus Infection	Asymmetric limb weakness, fever, no sensory loss	Supportive care, physical therapy, vaccination in community
Non-polio Enteroviruses	Similar to poliovirus, often with respiratory/gastrointestinal symptoms	Supportive care, symptomatic treatment

Guillain-Barré Syndrome (GBS)	Rapidly progressive limb weakness, areflexia, ascending paralysis	Hospitalization, IV immunoglobulins, plasmapheresis
Transverse Myelitis	Weakness, sensory alterations, autonomic dysfunction	Corticosteroids, immunosuppressive therapy, rehabilitation
Traumatic Neuritis	Localized weakness and pain, history of injury	Pain management, physical therapy
Tick Paralysis	Ascending paralysis, history of tick exposure	Tick removal, supportive care
Hypokalemic Periodic Paralysis	Episodes of muscle weakness, low potassium levels	Potassium supplements, avoiding triggers
Botulism	Descending paralysis, cranial nerve involvement	Antitoxin administration, supportive care
Spinal Muscular Atrophy	Muscle weakness and atrophy, genetic history	Supportive care, genetic counseling, nusinersen for some types
Myasthenia Gravis	Fluctuating muscle weakness, ocular symptoms	Acetylcholinesterase inhibitors, immunosuppressants, thymectomy
Toxic Neuropathies	Weakness, history of toxin/drug exposure	Removal of toxin, supportive care
Rabies	Progressive paralysis, history of animal bite	Post-exposure prophylaxis if not vaccinated, supportive care

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Q: Define criteria for declaring a country Polio free. What is the present status of wild polio virus transmission and strategies being used for its control in India?

Elaborate on AFP surveillance

Criteria for Declaring a Country Polio-Free

- **No Wild Poliovirus Cases:** No reported cases of wild poliovirus (WPV) for at least three consecutive years.

- **High-Quality Surveillance:** Effective acute flaccid paralysis (AFP) surveillance system in place, meeting specific performance indicators.
- **Laboratory Confirmation:** Absence of WPV isolation in laboratory tests from all AFP cases and from environmental samples.
- **Vaccination Coverage:** High immunization coverage with Oral Polio Vaccine (OPV) and/or Inactivated Polio Vaccine (IPV) in all regions.
- **Outbreak Preparedness:** Robust plans for rapid response to potential poliovirus importations or outbreaks.

Present Status of Wild Poliovirus Transmission in India

- **Polio-Free Certification:** India was declared polio-free by the World Health Organization (WHO) in 2014.
- **Last Case:** The last case of wild poliovirus in India was reported in 2011.
- **Vaccine-Derived Poliovirus (VDPV):** Surveillance for VDPV continues, as it can cause outbreaks in under-immunized communities.

Strategies for Polio Control in India

- **National Immunization Days (NIDs):** Regular mass immunization campaigns targeting all children under 5 years of age.
- **Pulse Polio Program:** Intensive efforts to administer OPV to all children, especially in high-risk areas.
- **Routine Immunization Strengthening:** Ensuring high coverage of polio vaccines in routine immunization schedules.
- **Surveillance Enhancement:** Strengthening AFP surveillance and environmental surveillance for poliovirus detection.
- **Cross-Border Collaboration:** Collaborating with neighbouring countries to prevent poliovirus importation.

AFP Surveillance

- **Definition:** AFP surveillance involves the detection and investigation of all cases of acute flaccid paralysis in children under 15 years of age.
- **Case Reporting:** Mandatory reporting of all AFP cases to health authorities.
- **Investigation:** Each reported case is investigated to determine the cause, with stool samples collected for laboratory analysis.

- **Laboratory Testing:** Testing for poliovirus in stool samples to confirm or rule out poliovirus as the cause of paralysis.
- **Performance Indicators:** Surveillance quality is assessed using indicators like non-polio AFP rate and stool adequacy rate.
- **Environmental Surveillance:** Complementing AFP surveillance by testing sewage samples for the presence of poliovirus.
- **Data Analysis:** Continuous analysis of surveillance data to guide immunization strategies and detect potential outbreaks.

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Q: Vaccine-Derived Paralytic Polio

Definition:

- **Vaccine-Derived Paralytic Polio (VDPP):** A rare condition caused by mutated strains of the poliovirus that are initially contained in the Oral Polio Vaccine (OPV).

How It Occurs:

- **Mutation:** The live, weakened virus in OPV can sometimes mutate in the gut and regain its ability to cause paralysis.
- **Transmission:** The mutated virus can spread in communities with low vaccination coverage, leading to VDPP cases.

Types of VDPP:

1. **Circulating Vaccine-Derived Poliovirus (cVDPV):** When the mutated virus spreads in the community.
2. **Immunodeficiency-Related Vaccine-Derived Poliovirus (iVDPV):** Occurs in individuals with immune system deficiencies.

Risk Factors:

- **Low Immunization Rates:** Communities with low vaccination coverage are at higher risk.
- **Immunodeficiency:** Individuals with compromised immune systems are more susceptible.

Prevention:

- **High Vaccination Coverage:** Ensuring high coverage with both OPV and IPV (Inactivated Polio Vaccine) in all communities.
- **Switch to IPV:** In some cases, transitioning from OPV to IPV to eliminate the risk of VDPP.
- **Surveillance:** Strengthening AFP and environmental surveillance to detect and respond to VDPP outbreaks.

Management of Outbreaks:

- **Rapid Response:** Immediate mass immunization campaigns with OPV and/or IPV in response to VDPP outbreaks.
- **Surveillance Intensification:** Enhanced AFP and environmental surveillance in affected areas.
- **Public Health Measures:** Education, sanitation, and hygiene measures to prevent virus spread.

Global Efforts:

- **Global Polio Eradication Initiative (GPEI):** Working towards the eradication of all polioviruses, including VDPP.
- **Vaccine Strategy:** Gradual phasing out of OPV and introduction of IPV in global immunization programs.

Q: Prevalence, clinical manifestations and management of Covid-19 infections in children

Prevalence of COVID-19 in Children

- **Lower Prevalence:** Initially, children represented a smaller proportion of COVID-19 cases compared to adults.
- **Increased Cases Over Time:** With the spread of new variants, there has been an increase in cases among children.
- **Milder Course:** Generally, children experience a milder course of the disease compared to adults.

Clinical Manifestations of COVID-19 in Children

- **Respiratory Symptoms:** Cough, sore throat, nasal congestion, and difficulty breathing.

- **Fever and Fatigue:** Common symptoms, though some children may be asymptomatic.
- **Gastrointestinal Symptoms:** Nausea, vomiting, diarrhea, and abdominal pain.
- **Skin Rashes:** Some children develop skin rashes or lesions.
- **Multisystem Inflammatory Syndrome in Children (MIS-C):** A rare but severe complication characterized by inflammation in multiple organs, including the heart, lungs, kidneys, brain, skin, eyes, or gastrointestinal organs.

Management of COVID-19 in Children

- **Mild Cases:**
 - Home Care: Rest, hydration, and management of symptoms.
 - Over-the-Counter Medications: For fever and pain relief.
 - Monitoring: Keep an eye on symptoms and seek medical attention if they worsen.
- **Moderate to Severe Cases:**
 - Hospitalization: May be required for severe cases or those with underlying health conditions.
 - Oxygen Therapy: For children with respiratory distress.
 - Medications: Antiviral drugs, steroids, or other treatments as per clinical guidelines.
 - Supportive Care: Fluid management, nutritional support, and treatment of any secondary infections.
- **Prevention:**
 - Vaccination: COVID-19 vaccines for eligible age groups.
 - Hygiene Measures: Handwashing, mask-wearing, and physical distancing.
 - Limiting Exposure: Avoiding crowded places and close contact with sick individuals.
- **MIS-C Management:**
 - Hospitalization: Often requires care in a pediatric intensive care unit (PICU).

- Medications: Intravenous immunoglobulin (IVIG), steroids, and other immunomodulatory therapies.
- Supportive Care: Treatment of symptoms and support for affected organ systems.

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Q: Epidemiology, clinical features, prognosis and prevention of Japanese Encephalitis

Epidemiology of Japanese Encephalitis (JE)

- **Geographical Distribution:** Predominantly found in rural and peri-urban areas of Southeast Asia, the Western Pacific, and parts of the Indian subcontinent.
- **Transmission:** Transmitted to humans through the bite of infected Culex mosquitoes, which breed in rice paddies and other water bodies.
- **Reservoir Hosts:** Pigs and wading birds serve as primary reservoirs for the JE virus.
- **Seasonal Incidence:** Typically peaks during and after the rainy season, when mosquito populations are high.

Clinical Features of Japanese Encephalitis

- **Incubation Period:** Usually 5-15 days post-infection.
- **Asymptomatic Infections:** Most JE virus infections are asymptomatic or result in mild symptoms.
- **Symptomatic Infections:** Can range from mild flu-like symptoms to severe neurological disease.
- **Neurological Symptoms:** High fever, headache, neck stiffness, disorientation, coma, seizures, spastic paralysis.
- **Encephalitis:** Inflammation of the brain, which is a hallmark of severe JE.

Prognosis of Japanese Encephalitis

- **Case Fatality Rate:** Approximately 20-30% among those with encephalitis.
- **Neurological Sequelae:** Up to 50% of survivors have persistent neurological or psychiatric sequelae, including cognitive impairment, motor deficits, and epilepsy.

- **Recovery:** Some patients recover fully, but the recovery process can be slow and difficult.

Prevention of Japanese Encephalitis

- **Vaccination:** The most effective way to prevent JE. Several safe and effective vaccines are available.

Japanese Encephalitis (JE) Vaccines

Types of JE Vaccines

1. **Inactivated Mouse Brain-Derived Vaccines:**
 - Example: JE-VAX (no longer produced).
 - Derived from virus grown in mouse brains, inactivated by formalin.
2. **Inactivated Vero Cell-Derived Vaccines:**
 - Example: IXIARO/JESPECT.
 - Produced by growing the virus in Vero cells, followed by inactivation.
3. **Live Attenuated Vaccines:**
 - Example: SA 14-14-2 vaccine.
 - Developed from a weakened strain of the JE virus.
4. **Recombinant Live Attenuated Vaccines:**
 - Example: ChimeriVax-JE.
 - Uses genetic engineering to combine JE virus antigens with another virus.

Efficacy and Effectiveness

- **High Efficacy:** Most JE vaccines have shown high efficacy in preventing JE in clinical trials.
- **Duration of Protection:** Generally provides long-term protection, but booster doses may be recommended.

Dosage and Administration

- **Dosage:** Varies depending on the vaccine. Some require a single dose, while others require multiple doses.
- **Booster Doses:** May be recommended for prolonged protection, especially for travelers or those in endemic areas.

Indications

- **Travelers:** Recommended for travelers to JE-endemic areas who will spend extensive time outdoors.
- **Residents:** Recommended for people living in endemic areas, especially in rural regions with high mosquito exposure.

Contraindications and Precautions

- **Allergies:** Contraindicated in individuals with severe allergic reactions to vaccine components.
- **Pregnancy and Breastfeeding:** Safety data may be limited; consult healthcare providers for recommendations.

Side Effects

- **Common Side Effects:** Pain at the injection site, mild fever, headache.
- **Severe Reactions:** Rare, but can include allergic reactions or neurological symptoms.

Storage and Handling

- **Cold Chain:** Most JE vaccines require refrigeration for storage.
- **Handling:** Should be administered by trained healthcare professionals.

Global Recommendations

- **WHO Recommendations:** The World Health Organization recommends JE vaccination in all regions where the disease is a recognized public health problem.

Public Health Impact

- **Reduction in Cases:** Widespread vaccination has significantly reduced JE cases in several countries.
- **Integration into National Immunization Programs:** Many endemic countries have integrated JE vaccines into their routine immunization schedules for children.

Other measures of prevention

- **Mosquito Control:** Reducing mosquito populations through source reduction (eliminating breeding sites), insecticide spraying, and use of larvicides.

- **Personal Protection:** Using mosquito repellents, wearing long-sleeved clothing, and using bed nets, especially during peak mosquito activity times.
- **Public Health Measures:** Surveillance, public awareness campaigns, and vaccination programs in endemic areas.
- **Travel Precautions:** Travelers to endemic areas should consider vaccination and take measures to avoid mosquito bites.

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Q: Clinical features and treatment of Chikungunya

Clinical Features of Chikungunya

Acute Phase (First Week)

- **Fever:** Sudden onset, high-grade, typically lasting 2-3 days.
- **Joint Pain:** Severe, often debilitating, affecting multiple joints symmetrically.
- **Rash:** Maculopapular or erythematous rash, usually appearing 2-5 days after fever onset.
- **Headache:** Common, along with other flu-like symptoms.
- **Muscle Pain:** Widespread myalgia.
- **Swelling:** Joint swelling and stiffness.
- **Other Symptoms:** Nausea, fatigue, conjunctivitis, and gastrointestinal complaints.

Subacute Phase (Weeks to Months)

- **Persistent Joint Pain:** Can last for weeks to months, sometimes years.
- **Recurrent Arthralgia:** Fluctuating intensity of joint pain.
- **Fatigue:** Lingering tiredness and malaise.

Chronic Phase (Months to Years)

- **Chronic Arthralgia:** In some cases, joint pain can become chronic.
- **Other Chronic Symptoms:** Depression, fatigue, and a reduced quality of life.

Treatment of Chikungunya

Symptomatic Management

- **Antipyretics:** Paracetamol (acetaminophen) for fever and pain relief.
- **Nonsteroidal Anti-Inflammatory Drugs (NSAIDs):** Ibuprofen or naproxen for joint pain, after acute fever phase.
- **Hydration:** Adequate fluid intake to prevent dehydration.
- **Rest:** Sufficient rest to aid recovery.

Joint Pain Management

- **Physical Therapy:** Rehabilitation exercises to improve joint function and reduce pain.
- **Analgesics:** Pain management as per the severity of symptoms.
- **Corticosteroids:** Short courses may be considered for severe, persistent arthralgia.

Supportive Care

- **Nutritional Support:** Balanced diet to support recovery.
- **Psychological Support:** Counseling for patients dealing with chronic pain and fatigue.

Prevention of Secondary Infections

- **Skin Care:** Proper care of skin lesions to prevent secondary bacterial infections.

Monitoring and Follow-Up

- **Regular Follow-Up:** Monitoring for the resolution of symptoms and management of chronic complications.

No Specific Antiviral Treatment

- **Note:** Currently, there is no specific antiviral medication for Chikungunya.

Prevention

- **Vector Control:** Measures to reduce mosquito populations and prevent bites.
- **Public Health Measures:** Education and awareness to prevent mosquito breeding and bites.

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Q: Define DHF and DSS ; Dengue Fever

DHF and DSS represent the severe spectrum of dengue fever

Dengue Hemorrhagic Fever (DHF)**Definition**

- ***Dengue Hemorrhagic Fever (DHF)***: A severe form of dengue fever characterized by bleeding, blood plasma leakage, and low platelet count.

Criteria for DHF

1. ***Fever***: High fever lasting 2-7 days.
2. ***Hemorrhagic Tendencies***: Evidence of bleeding (e.g., skin hemorrhages, nose or gum bleeding, internal bleeding).
3. ***Thrombocytopenia***: Low platelet count ($<100,000/\text{mm}^3$).
4. ***Plasma Leakage***: Signs of plasma leakage such as pleural effusion, ascites, or hypoproteinemia.

Dengue Shock Syndrome (DSS)**Definition**

- ***Dengue Shock Syndrome (DSS)***: The most severe form of dengue, characterized by severe plasma leakage leading to shock.

Criteria for DSS

1. ***All Criteria of DHF***: DSS includes all the criteria of DHF.
2. ***Circulatory Failure***: Signs of circulatory failure, such as rapid and weak pulse, hypotension, or cold, clammy skin.
3. ***Shock***: Evidence of circulatory shock, such as a narrow pulse pressure (<20 mm Hg) or hypotension for age.

Dengue Fever**Definition**

- ***Dengue Fever***: A viral illness caused by the dengue virus, transmitted by Aedes mosquitoes.

Clinical Features

- ***Sudden Onset Fever***: High fever that may last for 2-7 days.
- ***Severe Headache***: Especially behind the eyes.

- **Joint and Muscle Pain:** Often severe and debilitating.
- **Rash:** Skin rash that appears during the fever or after the fever subsides.
- **Nausea and Vomiting:** Common gastrointestinal symptoms.
- **Mild Bleeding:** Such as nose or gum bleeding, or easy bruising.

Management

- **Symptomatic Treatment:** Paracetamol for fever and pain, adequate hydration, and rest.
- **Monitoring:** Close monitoring of vital signs, hematocrit, and platelet count.
- **Hospitalization:** In cases of DHF or DSS, hospitalization for fluid management and supportive care is crucial.

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Q: Classification and Grading of Dengue Hemorrhagic Fever:

1. **Grade I:**

- Fever accompanied by non-specific constitutional symptoms.
- The only hemorrhagic manifestation is a positive tourniquet test.
- There may be no other signs of plasma leakage.

2. **Grade II:**

- Grade I manifestations plus spontaneous bleeding, usually in the form of skin hemorrhages (petechiae, purpura) or minor mucosal bleeding (nose or gums).
- No signs of shock.

3. **Grade III (Dengue Shock Syndrome DSS):**

- Signs of circulatory failure manifest.
- Rapid, weak pulse with narrowing of the pulse pressure (≤ 20 mm Hg) or hypotension with the presence of cold, clammy skin and restlessness.
- Evidence of plasma leakage such as pleural effusion or ascites.

4. **Grade IV (DSS):**

- Profound shock with undetectable blood pressure or pulse.

This is the most severe form and is life-threatening.Q: **The classification of dengue fever**

- ❖ Dengue, a mosquito-borne viral illness caused by the dengue virus, is crucial for appropriate clinical management and prognosis
- ❖ The World Health Organization (WHO) has provided guidelines for the classification of dengue fever, which are widely used globally
- ❖ The WHO 2009 classification of dengue fever into dengue without warning signs, dengue with warning signs, and severe dengue provides a framework for clinicians to assess the severity of the disease and make appropriate management decisions
- ❖ This classification emphasizes the identification of warning signs that may precede severe complications, thereby improving patient outcomes through timely intervention.

WHO 2009 Classification:

1. **Dengue without Warning Signs:**

- Fever and two or more of the following symptoms: nausea, vomiting, rash, aches and pains, leukopenia, positive tourniquet test.
- No signs of plasma leakage, fluid accumulation, respiratory distress, severe bleeding, or organ impairment.

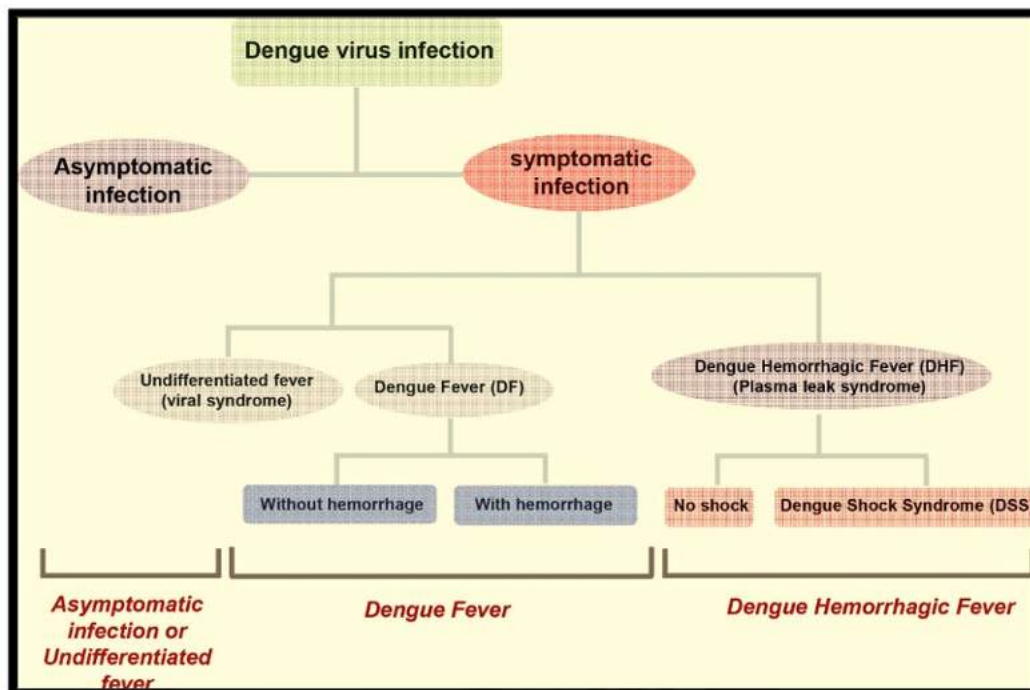
2. **Dengue with Warning Signs:**

- Abdominal pain or tenderness.
- Persistent vomiting.
- Clinical fluid accumulation (ascites, pleural effusion).
- Mucosal bleed.
- Lethargy or restlessness.
- Liver enlargement >2 cm.
- Increase in hematocrit concurrent with rapid decrease in platelet count.

3. **Severe Dengue:**

- Severe plasma leakage leading to shock (dengue shock syndrome) or fluid accumulation with respiratory distress.
- Severe bleeding as evaluated by clinician.

- Severe organ involvement such as liver (AST or ALT ≥ 1000 IU/L), CNS (impaired consciousness), or heart.



Additional Considerations:

- **Dengue Hemorrhagic Fever (DHF) and Dengue Shock Syndrome (DSS):** These terms were more commonly used in the older classification (prior to 2009) but are now encompassed under "Severe Dengue".
- **Laboratory Confirmation:** Diagnosis is confirmed through serological tests, PCR, or NS1 antigen detection.
- **Management:** The classification guides clinical management, from outpatient care for dengue without warning signs to hospitalization and intensive care for severe dengue.

Pediatric Considerations:

- **Warning Signs in Children:** Children may present with different warning signs, such as irritability or somnolence.
- **Shock:** Children, especially infants, are at higher risk for dengue shock syndrome.

Public Health Implications:

- **Epidemiological Surveillance:** Classification aids in monitoring and responding to dengue outbreaks.

- **Preventive Measures:** Vector control, public awareness, and vaccine research are integral to dengue prevention.

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Q: Outline the WHO criteria for diagnosis of dengue hemorrhagic fever. Draw an algorithm for volume replacement for a child with DHF and > 20% increase in hematocrit.

WHO Criteria for Diagnosis of Dengue Hemorrhagic Fever (DHF)

1. **Fever or History of Acute Fever:** Lasting 2-7 days, often biphasic.
2. **Hemorrhagic Manifestations:** At least one of the following:
 - Positive tourniquet test.
 - Petechiae, ecchymoses, or purpura.
 - Bleeding from mucosa, gastrointestinal tract, injection sites, or other locations.
 - Hematemesis or melena.
3. **Thrombocytopenia:** Platelet count $\leq 100,000/\text{mm}^3$.
4. **Evidence of Plasma Leakage:** Due to increased vascular permeability, indicated by at least one of the following:
 - A rise in hematocrit $\geq 20\%$ above baseline or after fluid replacement.
 - Signs of plasma leakage such as pleural effusion, ascites, or hypoproteinemia.

Volume Replacement in DHF with >20% Increase in Hematocrit:

1. **Initial Assessment:**
 - Evaluate vital signs and degree of haemoconcentration, dehydration, and electrolyte imbalance.
 - Monitor closely for at least 48 hours as shock may occur or recur precipitously, usually several days after the onset of fever.
2. **Oxygen Administration:**
 - Provide oxygen to patients who are cyanotic or have labored breathing.

3. **Fluid Replacement:**

- Rapid intravenous replacement of fluids and electrolytes is crucial.
- Normal saline is more effective than Ringer lactated saline in treating shock.
- Carefully monitor fluid administration to avoid overhydration, which may contribute to cardiac failure.

Fluid Management in DHF (Grades I and II):

a. Initial Management:

- Administer oxygen to all children (if not started yet).
- Infuse Ringer's lactate at a rate of 7 ml/kg over one hour.

b. Assessment After One Hour:

- If hematocrit decreases and vital parameters improve:
 - Decrease fluid infusion rate to 5 ml/kg over the next hour.
 - Further reduce to 3 ml/kg/hour for 24-48 hours.

c. Discharge Criteria:

- Normal blood pressure, satisfactory oral intake, and urine output indicate stability for discharge.

d. If No Improvement After One Hour:

- Increase fluid infusion rate to 10 ml/kg over the next hour.
- If still no improvement, further increase to 15 ml/kg over the third hour.

e. Use of Colloids or Plasma:

- If no improvement in vital parameters and hematocrit after 3 hours, administer colloids or plasma (10 ml/kg).

f. Stabilization and Discontinuation:

- Once hematocrit and vital parameters are stable, gradually reduce the infusion rate.
- Discontinue over 24-48 hours.

Fluid Management in DHF (Grades III and IV) and DSS:

a. Initial Management:

- Administer oxygen to all children with hypotension.
- Nasal Continuous Airway Pressure (NCPAP) may be a better option than oxygen by face mask, especially in respiratory failure.

b. Initial Resuscitation:

- Ringer's lactate solution, 10-20 ml/kg infused over one hour or given as a bolus of 20 ml/kg if blood pressure is unrecordable (DSS grade IV).
- The bolus may be repeated twice if there is no improvement.

c. Use of Colloids:

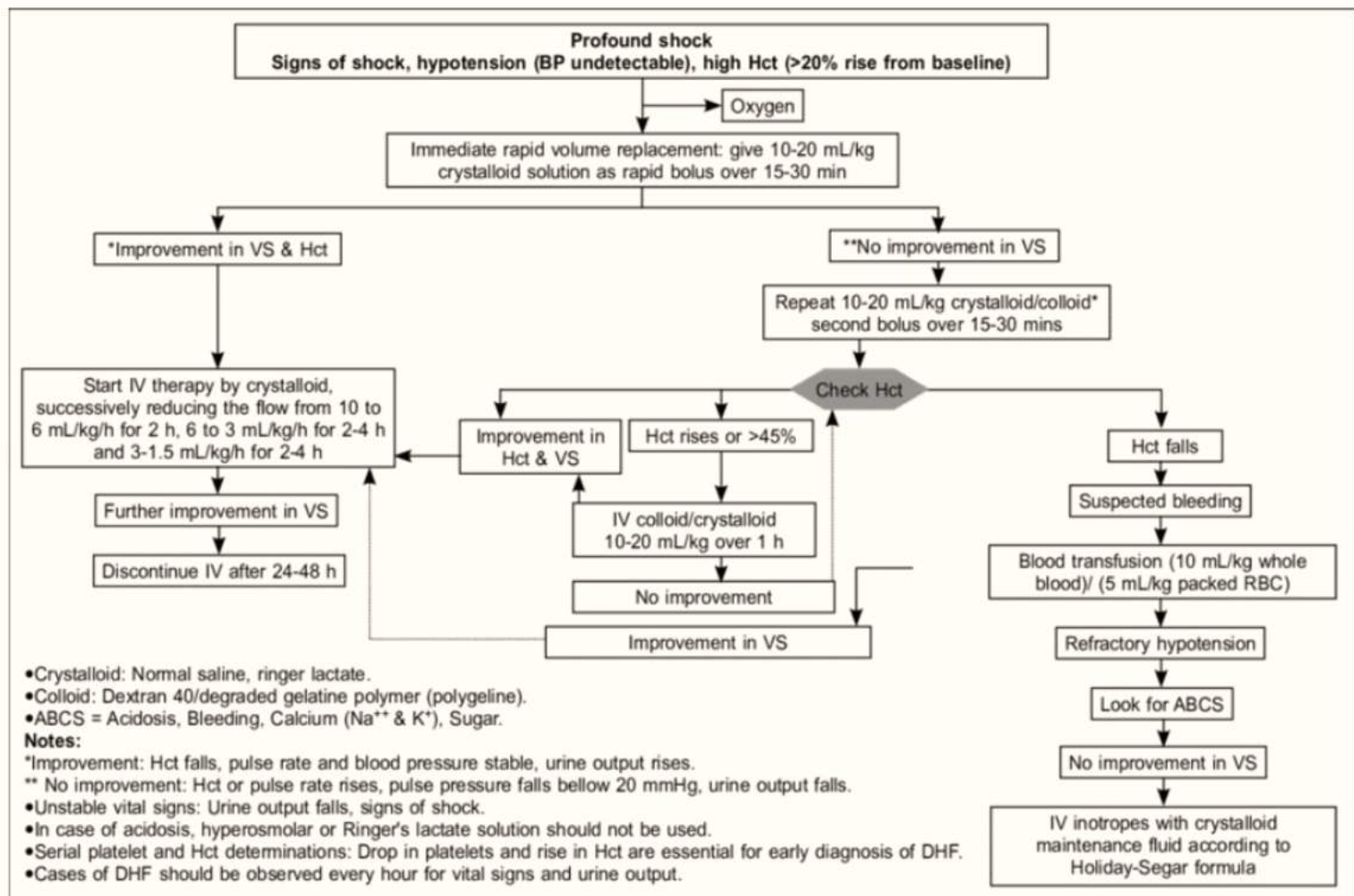
- If there is no improvement in vital parameters and hematocrit is rising, rapidly infuse colloids 10 ml/kg.

d. Blood Transfusion:

- If hematocrit is falling without improvement in vital parameters, transfuse blood, presuming occult blood loss.

e. Gradual Decrease:

- Once improvement starts, gradually decrease the fluid infusion rate.



4. Plasma or Colloid Preparations:

- Indicated when pulse pressure is ≥ 10 mm Hg or when elevation of the hematocrit persists after the replacement of fluids.

5. Oral Rehydration:

- Useful for children who are being monitored and can tolerate oral intake.

6. Avoidance of Certain Medications:

- Salicylates are contraindicated due to their effect on blood clotting.
- Avoid prophylactic platelet transfusions as they have not been shown to reduce the risk of hemorrhage or improve low platelet counts and may be associated with adverse effects.

7. **Management of Complications:**

- Hypervolemia during the fluid reabsorptive phase may be life-threatening, indicated by a decrease in hematocrit with wide pulse pressure.
- Diuretics and digitalization may be necessary.
- Transfusions of fresh blood may be required to control bleeding but should not be given during hemoconcentration but only after evaluation of hemoglobin or hematocrit values.

8. **Sedation:**

- May be required for children who are markedly agitated.

9. **Use of Vasopressors:**

- Has not resulted in a significant reduction of mortality rates over that observed with simple supportive therapy.

10. **Disseminated Intravascular Coagulation:**

- May require treatment

11. **Corticosteroids:**

- Do not shorten the duration of disease or improve the prognosis in children receiving careful supportive therapy.

12. **Prevention:**

- Dengue vaccines have been under development and one such vaccine, Dengvaxia, developed by Sanofi Pasteur, is available.

13. **Prognosis:**

- The prognosis of DHF is adversely affected by a late diagnosis and delayed or improper treatment.
- Death has occurred in 40-50% of patients with shock, but with adequate intensive care, deaths should occur in less than 1% of cases.

14. **Monitoring and Follow-up:**

Close monitoring is essential, especially during the critical phase of the illness.

Monitoring of children DHF/ DSS should include

1. Vital signs every 15-30 minutes till stable, then 1-2 Hourly

2. Hematocrit every 2 hr for first 6 hr, then every 4 hr till the patient is stable; Monitor 12 hourly during recovery.
3. Fluid balance sheet type of fluid, amount, rate etc.
4. Accurate urine Output
5. Serum electrolytes and blood gases every 12hrly
6. DIC profile and liver function tests as and when indicated.
7. Weight every 12hrly

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Q: Define severe dengue. Describe the WHO guidelines for its management.

Enumerate complications of Severe Dengue

Definition of Severe Dengue

Severe dengue, also known as dengue hemorrhagic fever or dengue shock syndrome, is a potentially life-threatening complication of dengue fever. It is characterized by:

- Plasma leakage leading to shock (dengue shock) and/or fluid accumulation with respiratory distress.
- Severe bleeding.
- Severe organ impairment (liver, CNS, heart).

WHO Guidelines for Management of Severe Dengue

1. Early Recognition and Treatment:

- Early identification of warning signs (abdominal pain, persistent vomiting, fluid accumulation, mucosal bleed, lethargy, liver enlargement, increasing hematocrit with decreasing platelets).
- Close monitoring of vital signs, hematocrit, and platelet count.

2. Fluid Management:

- Careful fluid management is crucial. Intravenous fluids are administered to maintain adequate circulation.
- Crystalloids are preferred for initial resuscitation. Colloids may be considered in cases of profound shock.
- Monitoring of fluid balance, hematocrit, and signs of fluid overload is essential.

3. **Management of Shock:**

- Prompt recognition and treatment of shock.
- Intravenous fluid therapy to restore plasma volume.
- Inotropic support in cases of refractory shock.

4. **Treatment of Severe Bleeding:**

- Transfusion of fresh whole blood or packed red cells for significant bleeding.
- Platelet transfusion may be considered in cases of severe thrombocytopenia with active bleeding.

5. **Organ Support:**

- Supportive care for organ dysfunction (renal, hepatic, cardiac, or neurological).

6. **Monitoring and Supportive Care:**

- Regular monitoring in an intensive care setting.
- Supportive care including pain management, treatment of fever, and management of other complications.

Complications of Severe Dengue

1. **Dengue Shock Syndrome (DSS):**

- Characterized by rapid and weak pulse, cold clammy skin, restlessness, and hypotension.

2. **Severe Hemorrhage:**

- Gastrointestinal bleeding, epistaxis, gum bleeding, or menorrhagia.

3. **Organ Impairment:**

- Hepatic dysfunction, acute liver failure.
- Renal impairment, acute kidney injury.
- Cardiac abnormalities.
- Neurological complications (encephalopathy, seizures).

4. **Fluid Accumulation and Respiratory Distress:**

- Pleural effusion, ascites.

- Respiratory distress due to fluid overload.

5. **Secondary Infections:**

- Increased risk of bacterial or other secondary infections due to immunosuppression.

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Q: Diagnostic lab tests for dengue fever

1. Virus Detection:

- **NS1 Antigen Test:** Detects dengue virus NS1 antigen. It's most useful within the first 1-7 days of illness. Rapid and can be performed at the point of care.
- **RT-PCR (Reverse Transcriptase Polymerase Chain Reaction):** Detects dengue virus RNA. Highly specific and sensitive, particularly in the early phase of the illness.

2. Serology:

- **IgM and IgG Antibody Tests:** Detects antibodies produced in response to dengue virus.
 - **IgM:** Becomes detectable 3-5 days after the onset of symptoms and indicates recent infection.
 - **IgG:** Appears later in the course of the infection and indicates past infection or secondary infection.

3. Complete Blood Count (CBC):

- **Thrombocytopenia:** Low platelet count is common in dengue fever.
- **Leukopenia:** Decreased white blood cell count.
- **Hematocrit:** Elevated hematocrit may indicate hemoconcentration, a warning sign for severe dengue.

4. Liver Function Tests:

- Elevated liver enzymes (AST and ALT) are often seen in dengue fever, indicating liver involvement.

5. Coagulation Profile:

- In severe cases, coagulation tests may show prolonged prothrombin time (PT) and activated partial thromboplastin time (aPTT), indicating coagulopathy.

6. Electrolyte Panel:

- Assessment of electrolyte balance, especially in severe cases with fluid loss or imbalance.

7. Urine Analysis:

- To check for proteinuria or hematuria, which can occur in severe cases.

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Q: What are the fluid, metabolic and biochemical changes in a child with severe dengue? Discuss the underlying pathophysiology

Severe dengue, also known as dengue hemorrhagic fever or dengue shock syndrome, can lead to significant fluid, metabolic, and biochemical changes in a child.

Severe dengue can lead to complex fluid, metabolic, and biochemical changes, primarily driven by increased vascular permeability and an exaggerated immune response.

Fluid Changes:

1. **Plasma Leakage:** The hallmark of severe dengue is increased vascular permeability leading to plasma leakage into the extravascular space. This results in hemoconcentration (increased hematocrit) and serous effusions (pleural effusion, ascites).
2. **Hypovolemia:** Plasma leakage causes intravascular volume depletion, leading to hypovolemia and potentially to dengue shock syndrome.
3. **Third Space Losses:** Fluid accumulates in the third space (interstitial space), which can lead to organ dysfunction due to edema.

Metabolic Changes:

1. **Metabolic Acidosis:** Hypovolemia and shock can lead to poor tissue perfusion and anaerobic metabolism, resulting in lactic acidosis and metabolic acidosis.
2. **Electrolyte Imbalances:** Dehydration and third space losses can lead to electrolyte imbalances, including hyponatremia and hypokalemia.

Biochemical Changes:

1. **Liver Dysfunction:** Elevated liver enzymes (AST, ALT) are common, and severe cases can lead to acute liver failure. This can affect the synthesis of clotting factors and glucose metabolism.

2. **Coagulopathy:** There may be a tendency towards bleeding due to thrombocytopenia (low platelet count), capillary fragility, and coagulation abnormalities.
3. **Altered Immune Response:** Cytokine storm and immune activation can lead to systemic inflammatory response and contribute to capillary leakage.

Underlying Pathophysiology:

1. **Viral Replication:** Dengue virus infects various cells, including monocytes and macrophages. The virus replicates and triggers an immune response.
2. **Immune Response:** The immune response to the dengue virus, especially during secondary infections, can be exaggerated, leading to a cytokine storm. This hyperactive immune response contributes to increased vascular permeability.
3. **Capillary Leakage:** The exact mechanism is not fully understood, but it's believed that cytokines and other mediators disrupt the endothelial cell barrier, leading to plasma leakage.
4. **Shock and Organ Dysfunction:** Hypovolemia and plasma leakage can lead to dengue shock syndrome. Poor perfusion and hypoxia can result in multi-organ dysfunction.

Q: Enumerate the indications for transfusion in dengue

Transfusion decisions in dengue should be made cautiously, considering the patient's clinical status, laboratory parameters, and the risk of fluid overload. Over-transfusion can exacerbate fluid overload and lead to complications.

Transfusion in dengue fever is a critical intervention, particularly in severe cases. Here are the indications for transfusion in dengue:

1. Platelet Transfusion:

- **Severe Thrombocytopenia:** Generally considered if platelet count drops below 10,000 to 20,000/mm³, especially if accompanied by active bleeding.
- **Significant Bleeding:** Regardless of platelet count, if there is active bleeding such as gastrointestinal bleeding, intracranial hemorrhage, or other severe hemorrhagic manifestations.

2. Red Blood Cell (RBC) Transfusion:

- **Significant Anemia:** Caused by bleeding or hemolysis.
- **Hemodynamic Instability:** In cases of dengue shock syndrome where there is evidence of poor tissue perfusion and oxygenation despite adequate fluid resuscitation.

3. Fresh Frozen Plasma (FFP) Transfusion:

- **Correction of Coagulopathy:** In cases with significant coagulation abnormalities, evidenced by prolonged prothrombin time (PT) or activated partial thromboplastin time (aPTT).
- **Severe Plasma Leakage:** To correct the imbalance in oncotic pressure due to severe plasma leakage.

4. Cryoprecipitate Transfusion:

- **Hypofibrinogenemia:** In cases where there is a significant decrease in fibrinogen levels.

5. Albumin Transfusion:

- **Severe Hypoalbuminemia:** Particularly in cases with significant third space losses and fluid refractory shock.

Q: Describe the etiology, mode of transmission, clinical features and management of viral hemorrhagic fever in children

Aspect	Details
Etiology	Arenaviridae (e.g., Lassa fever virus) ; Filoviridae (e.g., Ebola, Marburg viruses) ; Flaviviridae (e.g., Dengue, Yellow fever) ; Bunyaviridae (e.g., Hantavirus, Rift Valley fever virus) ; Typically zoonotic, transmitted from animals to humans
Mode of Transmission	Contact with infected animals or their excretions ; Bites from infected arthropods (mosquitoes, ticks) ; Exposure to body fluids of infected individuals ; Ingestion of contaminated food (e.g., Lassa fever)
Clinical Features	Fever, lethargy, headache, photophobia ; Nausea, vomiting, myalgia, arthralgia ; Abdominal pain, productive cough, mild respiratory distress ; Bleeding manifestations (epistaxis, hemoptysis, hematuria,

	hematemesis) ; Rash ; Complications: pneumonitis, meningoencephalitis, acute renal failure, myocarditis, septic shock-like syndrome
Management	Supportive care: hydration, management of fever and pain ; Treatment of secondary infections ; Specific antivirals for some VHF (e.g., Ribavirin for Lassa fever, Hantavirus) ; Blood products and intensive care for severe cases ; Prevention: avoid exposure to vectors and reservoirs, protective clothing, insect repellents

Additional Information:

- **Isolation and Infection Control:** Patients with VHF should be isolated, and strict infection control measures should be implemented to prevent nosocomial transmission.
- **Monitoring:** Close monitoring of vital signs, fluid balance, and laboratory parameters is essential.
- **Vaccination:** For some VHF, vaccines are available (e.g., Yellow fever vaccine).
- **Community Awareness and Education:** Especially in endemic areas, community education about the modes of transmission and preventive measures is crucial.

Q: Post exposure HIV prophylaxis

Post-exposure prophylaxis (PEP) for HIV involves taking antiretroviral medicines (ART) after being potentially exposed to HIV to prevent becoming infected. Here are the key aspects of PEP:

Timing:

- PEP should be started as soon as possible, ideally within 2 hours of exposure.
- It's most effective when started within 24 hours and should be initiated no later than 72 hours after exposure.

Indications:

- Occupational exposure: Needlestick injury or mucosal exposure to blood or body fluids from a known HIV-positive individual.
- Non-occupational exposure: Unprotected sexual intercourse, shared needles, or other exposures to potentially HIV-infected blood or body fluids.

Medication Regimen:

- A combination of three antiretroviral drugs is typically used.
- The regimen usually includes two nucleoside reverse transcriptase inhibitors (NRTIs) and a third drug from another class (often an integrase strand transfer inhibitor).
- Commonly used drugs include tenofovir, emtricitabine, and raltegravir or dolutegravir.

Duration:

- PEP should be taken daily for 28 days.

Monitoring and Follow-up:

- Baseline HIV testing should be done before starting PEP.
- Follow-up HIV testing is recommended at 6 weeks, 3 months, and 6 months post-exposure.
- Monitor for side effects and adherence to medication.

Side Effects:

- PEP can cause side effects, including nausea, diarrhea, fatigue, and headaches.
- Severe side effects are rare but require medical attention.

Counseling and Support:

- Counseling about the risk of HIV transmission, adherence to medication, and prevention of further exposure is essential.
- Psychological support may be needed, especially in cases of sexual assault.

Prevention:

- PEP is not a substitute for regular HIV prevention methods, such as safe sex practices and using clean needles.

Drug Name	Dosage	Common Side Effects
Tenofovir Disoproxil Fumarate (TDF)	300 mg once daily	Nausea, vomiting, diarrhea, headache, dizziness, kidney problems, bone loss
Emtricitabine (FTC)	200 mg once daily	Headache, diarrhea, nausea, fatigue, skin discoloration (especially on palms and soles)

Raltegravir (RAL)	400 mg twice daily	Insomnia, headache, dizziness, nausea, increased liver enzymes
Dolutegravir (DTG)	50 mg once daily	Insomnia, headache, diarrhea, nausea, increased liver enzymes
Lamivudine (3TC)	300 mg once daily	Fatigue, headache, nausea, diarrhea, cough, nasal symptoms
Zidovudine (AZT)	300 mg twice daily	Anemia, neutropenia, nausea, headache, muscle pain, darkening of nails and skin
Lopinavir/Ritonavir (LPV/r)	400/100 mg twice daily	Diarrhea, nausea, vomiting, headache, abdominal pain, increased liver enzymes, lipid abnormalities

Notes:

- The choice of drugs and dosage may vary based on individual circumstances, local guidelines, and the source patient's HIV status.
- Some side effects may be more severe and require medical attention.
- It's important to monitor kidney function and liver enzymes during PEP, especially with certain drugs like Tenofovir and Raltegravir.

Standard PEP Regimen:

1. **Tenofovir (TDF) + Lamivudine (3TC) + Dolutegravir (DTG)**
 - Tenofovir: 300 mg once daily
 - Lamivudine: 300 mg once daily
 - Dolutegravir: 50 mg once daily

Duration:

- The PEP regimen should be taken daily for a duration of 28 days.

Timing:

- PEP should be initiated as soon as possible after exposure, ideally within 2 hours, and no later than 72 hours.

Monitoring and Follow-up:

- Baseline HIV testing before starting PEP.
- Follow-up HIV testing at 6 weeks, 3 months, and 6 months post-exposure.

- Monitor for side effects and adherence to medication.

Counseling and Support:

- Counseling regarding the risk of HIV transmission, adherence to medication, and prevention of further exposure is crucial.
- Psychological support may be necessary, especially in cases of sexual assault.

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Q: Factors involved in perinatal transmission of HIV infection and the various preventive measures; Discuss the risk factors for vertical transmission of HIV infection and methods to prevent parent to child transmission of HIV.

The risk of perinatal transmission of HIV can be significantly reduced with appropriate interventions.

Factors Involved in Perinatal Transmission of HIV	Preventive Measures
<i>Maternal Factors</i>	<i>Interventions for Mother</i>
High maternal viral load	Initiate or continue Antiretroviral Therapy (ART) to achieve viral suppression
Low CD4 count	Regular monitoring of CD4 count and viral load
Advanced stage of HIV/AIDS	Treat any co-infections and maintain overall health
Untreated or inadequately treated HIV during pregnancy	Ensure adherence to ART regimen
<i>Obstetric Factors</i>	<i>Interventions during Labor and Delivery</i>
Mode of delivery (higher risk with vaginal delivery if viral load is high)	Elective Cesarean section if viral load is high or unknown
Prolonged rupture of membranes	Minimize duration of ruptured membranes
Invasive procedures during labor (e.g., fetal scalp electrodes)	Avoid unnecessary invasive procedures
<i>Infant Factors</i>	<i>Interventions for Newborn</i>

Prematurity	Administer neonatal antiretroviral prophylaxis as soon as possible after birth
Breastfeeding (if mother is not on effective ART)	Exclusive breastfeeding if mother is on effective ART; otherwise, use formula feeding
	Regular follow-up and HIV testing for the infant
Other Factors	General Measures
Co-infections (e.g., Hepatitis C, STIs)	Screen and treat co-infections
Substance abuse	Provide support for substance abuse treatment
Lack of prenatal care	Ensure access to and utilization of prenatal care services
Socioeconomic factors	Address social determinants of health, provide support and counseling

Q: An HIV positive mother has been admitted in labour. What will you do to prevent transmission of infection to the baby

Preventing mother-to-child transmission (MTCT) now called PPTCT, of HIV during labor and delivery involves a combination of medical interventions and supportive care.

The goal is to minimize the risk of HIV transmission while ensuring the health and well-being of both the mother and the baby.

1. Antiretroviral Therapy (ART) for the Mother:

- Continue or initiate ART for the mother, if not already on treatment.
- Ensure optimal viral suppression to reduce the risk of transmission.

2. Mode of Delivery:

- **Vaginal Delivery:** Generally recommended if the mother's viral load is well controlled.

- **Cesarean Section:** Considered if the viral load is high or unknown, especially close to delivery.

3. Intrapartum Antiretroviral Prophylaxis:

- Administer intravenous Zidovudine (AZT) to the mother during labor if her viral load is not well controlled.

4. Management During Labor:

- Avoid procedures that may increase the risk of MTCT, such as fetal scalp electrodes or episiotomy.
- Minimize the duration of ruptured membranes (breaking of water).

5. Postnatal Care for the Infant:

- Begin neonatal antiretroviral prophylaxis as soon as possible, preferably within 12 hours of birth.
- Commonly used regimen:
 - Nevirapine is particularly used when there is a high risk of MTCT, such as when the mother's viral load is high, or she has not received adequate antiretroviral therapy (ART) during pregnancy.

Dosage:

- The newborn should receive a single dose of Nevirapine syrup (2 mg/kg) within 72 hours of birth, ideally as soon as possible after delivery.

Duration:

- The single dose is typically followed by additional antiretroviral prophylaxis, such as Zidovudine (AZT), for 4-6 weeks.
- Oral Zidovudine for 4-6 weeks.
- Additional drugs may be added if the risk of transmission is high.

6. Feeding Recommendations:

- Exclusive breastfeeding is recommended if the mother is on effective ART and breastfeeding is culturally acceptable.
- If breastfeeding is not safe or feasible, use formula feeding.

7. Monitoring and Follow-up:

- Regular follow-up and HIV testing for the infant at recommended intervals (e.g., at birth, 6 weeks, 3 months, and 6 months).
- Monitor the infant for side effects of prophylaxis and signs of HIV infection.

8. Support and Counseling:

- Provide counseling and support to the mother regarding ART adherence, infant care, and feeding options.

9. Infection Control:

- Standard precautions to prevent exposure to HIV during delivery.

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Q: Prevention of HIV infection during childhood

Preventing HIV infection during childhood involves a multifaceted approach that includes medical interventions, education, and supportive measures. Here's a structured overview:

1. Prevention of Mother-to-Child Transmission (PMTCT) Now PPTCT:

- **Antiretroviral Therapy (ART) for Pregnant Women:** HIV-positive pregnant women should receive ART to reduce viral load and minimize the risk of transmission to the baby.
- **Safe Delivery Practices:** Elective Cesarean section if maternal viral load is high; minimize invasive procedures during labor.
- **Neonatal Prophylaxis:** Administer antiretroviral drugs to the newborn, typically within 12 hours of birth.

2. Feeding Practices:

- **Breastfeeding:** Exclusive breastfeeding is recommended if the mother is on effective ART. Otherwise, formula feeding may be advised to prevent transmission through breast milk.
- **Education on Safe Feeding:** Educate HIV-positive mothers on safe feeding practices to reduce the risk of transmission.

3. Vaccination and Health Maintenance:

- **Routine Vaccinations:** Ensure that children, especially those born to HIV-positive mothers, receive all routine vaccinations.

- **Regular Health Check-ups:** Monitor the child's health and development, and provide necessary medical care.

4. Education and Awareness:

- **HIV Education:** Educate children, as they grow older, about HIV transmission and prevention.
- **Sexual Health Education:** Provide age-appropriate sexual health education to prevent HIV transmission during adolescence.

5. Safe Practices and Injury Prevention:

- **Avoid Sharing Sharp Objects:** Educate children and caregivers about not sharing items like needles, razors, or toothbrushes.
- **First Aid and Wound Care:** Proper care of cuts and wounds to prevent exposure to blood-borne pathogens.

6. Support for Affected Families:

- **Psychosocial Support:** Provide support to children living with or affected by HIV and their families.
- **Nutritional Support:** Ensure adequate nutrition for children, particularly those living with HIV.

7. Prevention of Opportunistic Infections:

- **Prophylactic Medications:** For children living with HIV, provide prophylactic medications to prevent opportunistic infections.
- **Regular Monitoring:** Monitor for signs of opportunistic infections and provide timely treatment.

8. Access to Healthcare Services:

- **Healthcare Access:** Ensure access to healthcare services, including HIV testing and treatment.
- **Confidentiality and Stigma Reduction:** Maintain confidentiality and work towards reducing stigma associated with HIV.

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Q: Clinical Presentations requiring screening for HIV

HIV screening should be considered in a wide range of clinical scenarios, especially those associated with higher risk factors or unexplained clinical symptoms. Early identification through screening allows for timely intervention, management, and prevention of HIV transmission. It's important for healthcare providers to maintain a high index of suspicion and offer HIV testing as part of routine care in these clinical presentations.

Screening for HIV is an essential component of healthcare, especially in clinical scenarios that may indicate a higher risk of HIV infection. Here are some clinical presentations that warrant HIV screening:

1. Sexual Health-Related Presentations:

- **Sexually Transmitted Infections (STIs):** Diagnosis of any STI.
- **Multiple Sexual Partners:** Patients reporting multiple or anonymous sexual partners.
- **Unprotected Sexual Activity:** Engaging in unprotected sex, especially with partners of unknown HIV status.
- **Sexual Assault:** Victims of sexual assault.

2. Substance Use-Related Presentations:

- **Injection Drug Use:** Sharing needles or other injection equipment.
- **Substance Abuse:** Engaging in high-risk behaviors due to substance abuse.

3. Pregnancy and Perinatal Care:

- **Pregnant Women:** Routine screening during pregnancy.
- **Newborns of HIV-Positive Mothers:** Screening of newborns born to HIV-positive mothers.

4. Blood Exposure-Related Presentations:

- **Occupational Exposure:** Healthcare workers after needlestick injuries or exposure to potentially infected bodily fluids.
- **Non-Occupational Exposure:** Accidental exposure to needles or other sharp objects.

5. Clinical Symptoms and Conditions:

- **Unexplained Weight Loss:** Significant weight loss without a clear cause.
- **Persistent Fever:** Prolonged fever of unknown origin.

- **Recurrent Infections:** Frequent or unusual infections.
- **Chronic Diarrhea:** Persistent or unexplained diarrhea.
- **Oral Thrush:** Recurrent or persistent oral candidiasis.
- **Lymphadenopathy:** Unexplained swelling of lymph nodes.
- **Skin Rashes:** Unexplained persistent skin rashes or lesions.
- **Neurological Symptoms:** Unexplained neurological symptoms or cognitive changes.

6. Other Indications:

- **Blood Transfusion Recipients:** Especially if received before widespread screening of blood products.
- **Organ Transplant Recipients:** Particularly if the transplant occurred before routine donor screening.
- **Hemodialysis Patients:** Due to potential exposure to blood.
- **Tuberculosis (TB):** All patients diagnosed with TB should be screened for HIV.

7. High Prevalence Settings:

- **Geographic Prevalence:** In areas with high HIV prevalence, routine screening may be recommended for all patients.

Q: Outline clinical and immunological criteria for starting anti-retroviral treatment (ART) in a HIV infected child. How will you monitor a child initiated on ART?

Clinical Criteria for Initiating ART:

1. **Symptomatic Children:** Any HIV-infected child with symptoms, regardless of CD4 count or percentage.
2. **Asymptomatic Children with Low CD4 Counts:** Initiate ART in asymptomatic children based on age-specific CD4 count thresholds:
 - **Age < 12 months:** Any CD4 count.
 - **Age 12-35 months:** CD4 count <750 cells/mm³ or CD4 percentage <25%.
 - **Age 36-59 months:** CD4 count <350 cells/mm³ or CD4 percentage <20%.

- **Age $\geq$ 5 years:** CD4 count <350 cells/mm³.

Immunological Criteria for Initiating ART:

1. **CD4 Count and Percentage:** As mentioned above, specific CD4 count and percentage thresholds based on age are used to determine when to initiate ART in asymptomatic children.

Monitoring a Child Initiated on ART:

1. **Clinical Monitoring:**

- **Regular Follow-up Visits:** Schedule visits every 3-6 months.
- **Growth Monitoring:** Track weight, height, and developmental milestones.
- **Symptom Assessment:** Monitor for any new symptoms or worsening of existing conditions.
- **Adherence Counseling:** Ensure adherence to ART and address any barriers.

2. **Immunological Monitoring:**

- **CD4 Count:** Monitor CD4 count every 6 months to assess immune recovery.

3. **Virological Monitoring:**

- **Viral Load Testing:** Ideally, viral load should be monitored every 6 months to assess the effectiveness of ART. The goal is to achieve and maintain an undetectable viral load.

4. **Laboratory Monitoring:**

- **Complete Blood Count (CBC):** Monitor for anemia, neutropenia, or other hematological abnormalities.
- **Liver and Renal Function Tests:** Monitor liver enzymes and renal function periodically.
- **Lipid Profile:** Monitor lipid levels, especially in children on protease inhibitors.

5. **Management of Side Effects:**

- **Monitor for Adverse Drug Reactions:** Regularly assess for any side effects or toxicities related to ART and manage accordingly.

6. *Prevention and Treatment of Opportunistic Infections:*

- **Prophylaxis:** Provide prophylaxis for opportunistic infections as indicated.
- **Prompt Treatment:** Treat any opportunistic infections that may arise.

7. *Nutritional Support and Counseling:*

- **Nutritional Assessment:** Ensure the child is receiving adequate nutrition.
- **Dietary Counseling:** Provide guidance on a healthy diet.

8. *Psychosocial Support:*

- **Counseling Services:** Offer counseling for the child and family to address psychosocial issues.
- **Support Groups:** Connect the family with support groups if available.

Q: Enlist the common opportunistic infections in HIV infected children; Describe their clinical features, diagnosis and management

Opportunistic Infection	Clinical Features	Diagnosis	Management
<i>Pneumocystis jirovecii Pneumonia (PCP)</i>	Dyspnea, tachypnea, fever, cough	Chest X-ray: diffuse interstitial infiltrates; Induced sputum or bronchoalveolar lavage for microscopy and staining	Treatment: Trimethoprim-sulfamethoxazole (TMP-SMX); Prophylaxis: TMP-SMX in high-risk children
<i>Candidiasis (Oral/Esophageal)</i>	Oral: White plaques on oral mucosa; Esophageal: Dysphagia, pain on swallowing	Clinical diagnosis for oral; Endoscopy with biopsy for esophageal	Oral: Topical antifungals (e.g., nystatin); Esophageal: Systemic antifungals (e.g., fluconazole)

<i>Tuberculosis (TB)</i>	Persistent cough, fever, weight loss, night sweats	Sputum smear and culture; Chest X-ray; Tuberculin skin test or IGRA	Standard anti-TB therapy; ART should be started or continued
<i>Cytomegalovirus (CMV) Retinitis</i>	Visual changes, floaters, blindness	Ophthalmologic examination; PCR for CMV DNA	Ganciclovir or valganciclovir
<i>Toxoplasmosis</i>	Neurological symptoms: seizures, focal deficits	MRI or CT scan of brain; Serology for Toxoplasma antibodies	Pyrimethamine, sulfadiazine, and folinic acid
<i>Cryptococcal Meningitis</i>	Headache, fever, neck stiffness, photophobia	Lumbar puncture: CSF analysis; Cryptococcal antigen testing	Amphotericin B followed by fluconazole
<i>Mycobacterium avium Complex (MAC)</i>	Fever, weight loss, abdominal pain, anemia	Blood culture; Biopsy of affected tissues	Combination of macrolide (e.g., azithromycin) and other antibiotics (e.g., ethambutol)
<i>Herpes Simplex Virus (HSV) Infection</i>	Oral/genital ulcers, skin lesions	Clinical diagnosis; PCR or culture from lesions	Acyclovir or valacyclovir
<i>Varicella-Zoster Virus (VZV) Infection</i>	Painful vesicular rash, can be disseminated	Clinical diagnosis; PCR or culture from lesions	Acyclovir

- **Prophylaxis:** For certain infections like PCP and MAC, prophylactic treatment is recommended in high-risk children.
- **ART:** Antiretroviral therapy should be optimized in all HIV-infected children to improve immune function and reduce the risk of opportunistic infections.
- **Vaccination:** Ensure up-to-date vaccinations, including pneumococcal and Haemophilus influenzae type b vaccines, to prevent bacterial infections.
- **Nutritional Support:** Adequate nutrition is crucial for maintaining immune function.
- **Regular Monitoring:** Regular follow-up and monitoring for signs of opportunistic infections are essential.

Q: Describe the clinical features, diagnosis and management of herpes simplex infection in HIV infected children

Clinical Features:

1. **Oral Lesions:**

- Painful ulcers or blisters on the lips, tongue, gums, or inside the cheeks.
- Can be recurrent and more severe in HIV-infected children.

2. **Genital Lesions:**

- Painful ulcers or blisters on the genital or anal area.
- May be accompanied by itching or burning sensation.

3. **Skin Lesions:**

- Vesicular rash that can occur anywhere on the body.
- Lesions can be more extensive and persistent in HIV-infected children.

4. **Systemic Symptoms:**

- Fever, malaise, and lymphadenopathy, especially during the primary infection.

5. **Disseminated Infection:**

- In severe cases, HSV can disseminate, leading to more serious conditions like encephalitis, hepatitis, or pneumonitis.

Diagnosis:

1. **Clinical Diagnosis:**

- Often based on the characteristic appearance of lesions.

2. **Laboratory Tests:**

- **PCR (Polymerase Chain Reaction):** Highly sensitive and specific for detecting HSV DNA from lesion swabs, blood, or cerebrospinal fluid (CSF).
- **Viral Culture:** Can be used to isolate the virus from lesion swabs.
- **Serology:** Not typically used for acute diagnosis but can indicate past exposure to HSV.

3. **Imaging:**

- In cases of suspected disseminated infection or encephalitis, imaging studies like MRI or CT scan may be necessary.

Management:

1. Antiviral Therapy:

- **Acyclovir:** First-line treatment for HSV infections. Dosage may vary based on the severity and site of infection.
- **Valacyclovir or Famciclovir:** Alternatives, especially for older children who can take oral medication.

2. Topical Treatment:

- For mild oral or skin lesions, topical antiviral creams may be used.

3. Treatment of Complications:

- In cases of disseminated infection or encephalitis, hospitalization and intravenous antiviral therapy are necessary.

4. Pain Management:

- Analgesics for pain relief, especially for painful oral or genital lesions.

5. Supportive Care:

- Adequate hydration and nutritional support.
- Good oral hygiene for oral lesions.

6. Prevention of Recurrences:

- In children with frequent recurrences, suppressive antiviral therapy may be considered.

7. Optimizing ART:

- Ensure effective antiretroviral therapy to improve immune function and reduce the frequency of HSV recurrences.

8. Monitoring and Follow-up:

- Regular monitoring for signs of recurrence or complications.

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Q: In HIV infected children, How will you treat and prevent pneumocystis jirovecii infection? Describe the treatment and prophylactic regimes

Pneumocystis jirovecii pneumonia (PCP) is a serious opportunistic infection in HIV-infected children.

Treatment of Pneumocystis jirovecii Infection:

1. First-Line Therapy:

- **Trimethoprim-Sulfamethoxazole (TMP-SMX):**
 - Standard treatment for PCP.
 - Dosage: TMP 15-20 mg/kg/day and SMX 75-100 mg/kg/day, given in divided doses every 6 hours.
 - Duration: Typically 21 days.

2. Supportive Care:

- Oxygen therapy for hypoxemia.
- Adequate hydration and nutritional support.
- Management of any concurrent infections.

3. Adjunctive Therapy:

- **Corticosteroids:** Indicated in moderate to severe PCP (e.g., PaO₂ <70 mmHg on room air or alveolar-arterial gradient >35 mmHg).
 - Dosage: Prednisone or prednisolone at 1-2 mg/kg/day (maximum 40 mg/day) for 5-7 days, then taper over the next 7-14 days.

4. Monitoring and Follow-up:

- Regular monitoring of clinical response, oxygenation, and potential side effects of treatment.
- Follow-up chest radiographs may be necessary.

Prophylaxis of Pneumocystis jirovecii Infection:

1. Indications for Prophylaxis:

- In HIV-infected children with CD4 counts below age-specific thresholds or a history of an AIDS-defining illness.

2. First-Line Prophylactic Regimen:

- **Trimethoprim-Sulfamethoxazole (TMP-SMX):**

- Dosage for prophylaxis: TMP 150 mg/m²/day and SMX 750 mg/m²/day, given orally in divided doses twice a day, three times a week.
- Duration: Continues until immune reconstitution with effective antiretroviral therapy (ART).

3. **Alternative Prophylactic Regimens:**

- For children who cannot tolerate TMP-SMX:
 - **Dapsone:** Dosage based on age and weight.
 - **Atovaquone:** Oral suspension, dosage based on weight.
 - **Pentamidine:** Administered via inhalation, dosage based on weight.

4. **Monitoring:**

- Regular monitoring for side effects of prophylactic medications.
- Periodic reassessment of the need for prophylaxis based on CD4 counts and immune status.

Q: Discuss the pulmonary disorders seen in children with HIV/AIDS

Children with HIV/AIDS are at increased risk for a variety of pulmonary disorders. These disorders can be due to the direct effects of the HIV virus, opportunistic infections, or complications of the disease and its treatment.

1. Opportunistic Infections:

- ***Pneumocystis jirovecii* Pneumonia (PCP):**

- A common and potentially life-threatening infection.
- Presents with fever, cough, and difficulty breathing.
- Requires prompt treatment with Trimethoprim-Sulfamethoxazole (TMP-SMX).

- **Bacterial Pneumonia:**

- Increased susceptibility to bacterial pathogens like *Streptococcus pneumoniae* and *Haemophilus influenzae*.

- Presents with fever, cough, and respiratory distress.
- ***Tuberculosis (TB):***
 - Higher risk and more severe presentations.
 - Can present with chronic cough, weight loss, fever, and night sweats.
- ***Fungal Infections:***
 - Infections like histoplasmosis and coccidioidomycosis can occur, especially in endemic areas.

2. Non-Infectious Pulmonary Disorders:

- ***Lymphocytic Interstitial Pneumonitis (LIP):***
 - A chronic lung disease more common in HIV-infected children.
 - Presents with cough, dyspnea, and hypoxemia.
- ***Pulmonary Hypertension:***
 - Can be a complication of chronic lung disease or direct effect of HIV.

3. HIV-Associated Lung Disorders:

- ***HIV-Associated Bronchiectasis:***
 - Damage and dilation of the bronchial tubes, leading to chronic cough and recurrent infections.
- ***HIV-Associated Interstitial Lung Disease:***
 - A group of conditions causing scarring and inflammation of the lung tissue.

4. Complications of Antiretroviral Therapy (ART):

- ***Drug-Induced Lung Injury:***
 - Some antiretroviral drugs can cause lung problems like hypersensitivity reactions or pneumonitis.

5. Neoplasms:

- ***Lymphoma:***
 - Increased risk of lymphomas, which can involve the lungs.

6. Miscellaneous:

- ***Recurrent Respiratory Tract Infections:***

- Due to immune compromise, there's an increased frequency of respiratory infections.
- **Chronic Obstructive Pulmonary Disease (COPD):**
 - Long-term HIV infection and smoking can increase the risk of COPD.

Management:

- **Prompt Diagnosis and Treatment:**
 - Early recognition and treatment of pulmonary infections are crucial.
 - Regular monitoring for respiratory symptoms.
- **Prophylaxis:**
 - Prophylactic medications for opportunistic infections like PCP in high-risk children.
- **Optimizing ART:**
 - Effective ART to improve immune function and reduce the risk of opportunistic infections.
- **Supportive Care:**
 - Oxygen therapy, bronchodilators, and physiotherapy as needed.
- **Vaccinations:**
 - Up-to-date vaccinations, including pneumococcal and influenza vaccines.
- **Lifestyle Modifications:**
 - Avoidance of smoking and exposure to pollutants.

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Q: AIDS defining diseases in children

The presence of any of these conditions in an HIV-infected child is indicative of AIDS and requires immediate medical attention. Management typically involves aggressive treatment of the opportunistic infection or cancer, along with antiretroviral therapy to control HIV.

AIDS-defining illnesses are conditions that, in the presence of HIV infection, signify the progression from HIV infection to Acquired Immunodeficiency Syndrome (AIDS).

In children, these illnesses are particularly important as they indicate severe immunosuppression and the need for immediate and aggressive management.

1. Opportunistic Infections:

- ***Pneumocystis jirovecii Pneumonia (PCP)***
- ***Cytomegalovirus (CMV) Infections***: Retinitis, colitis, or encephalitis.
- ***Candidiasis***: Esophageal, tracheal, bronchial, or pulmonary.
- ***Cryptococcosis***: Extrapulmonary, such as cryptococcal meningitis.
- ***Cryptosporidiosis***: Chronic intestinal (lasting more than 1 month).
- ***Toxoplasmosis***: Brain toxoplasmosis.
- ***Mycobacterium avium Complex (MAC)***: Disseminated or extrapulmonary.
- ***Tuberculosis (TB)***: Pulmonary, disseminated, or extrapulmonary.
- ***Histoplasmosis***: Disseminated or extrapulmonary.

2. Viral Infections:

- ***Herpes Simplex Virus (HSV)***: Chronic ulcers (lasting more than 1 month) or bronchitis, pneumonitis, or esophagitis.
- ***Progressive Multifocal Leukoencephalopathy (PML)***

3. Fungal Infections:

- ***Coccidioidomycosis***: Disseminated or extrapulmonary.
- ***Histoplasmosis***: Disseminated or extrapulmonary.

4. Parasitic Infections:

- ***Isosporiasis***: Chronic intestinal (lasting more than 1 month).

5. Cancers:

- ***Kaposi's Sarcoma***
- ***Lymphoma***: Primary lymphoma of the brain, Burkitt's lymphoma, immunoblastic lymphoma, or other types of non-Hodgkin's lymphoma.
- ***Invasive Cervical Cancer***

6. Other Conditions:

- ***HIV Encephalopathy***: Also known as AIDS dementia complex.
- ***Wasting Syndrome***: Due to HIV.

- **Recurrent Bacterial Infections:** Multiple or recurrent.
- **Recurrent Pneumonia**

7. Pediatric-Specific Conditions:

- **Lymphoid Interstitial Pneumonitis (LIP)**
- **Recurrent Severe Bacterial Infections:** Such as empyema, pyomyositis, bone or joint infections, meningitis, or bacteremia.

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Q: WHO guidelines for breastfeeding in a baby born to HIV positive mother

The World Health Organization (WHO) provides guidelines for breastfeeding in infants born to HIV-positive mothers. These guidelines aim to balance the risk of HIV transmission through breast milk with the benefits of breastfeeding, especially in resource-limited settings where the risk of infant mortality and morbidity from other causes is high.

WHO Guidelines for Breastfeeding in Infants Born to HIV-Positive Mothers:

1. **Exclusive Breastfeeding:**
 - WHO recommends exclusive breastfeeding for the first 6 months of life, even for infants born to HIV-positive mothers. This means the infant should receive only breast milk and no other foods or liquids, not even water.
2. **Antiretroviral Therapy (ART):**
 - The mother should be on effective antiretroviral therapy (ART) to reduce the viral load. A suppressed viral load significantly reduces the risk of HIV transmission through breast milk.
 - Infants born to HIV-positive mothers should also receive antiretroviral prophylaxis to reduce the risk of HIV transmission.
3. **Mixed Feeding:**
 - Mixed feeding (breast milk along with other foods and liquids) before 6 months of age should be avoided as it increases the risk of HIV transmission.
4. **Continued Breastfeeding:**

- After 6 months, in addition to breast milk, complementary foods should be introduced. WHO recommends continued breastfeeding up to 12 months and beyond if the mother and baby are adherent to ART and the mother's viral load is undetectable or very low.

5. **Regular Monitoring:**

- Regular monitoring of the mother's health and viral load is important. The infant should also be regularly tested for HIV according to local guidelines.

6. **Counseling and Support:**

- Mothers should receive counseling about the benefits and risks of breastfeeding, and support in making an informed decision. They should also receive support to adhere to ART and maintain a healthy lifestyle.

7. **Safe Alternatives:**

- In settings where safe and affordable alternatives to breastfeeding are available, and the mother's health and viral load are not well controlled, the use of formula or other breast milk substitutes may be recommended.

8. **Nutrition and Health:**

- The health and nutritional status of both the mother and the infant should be monitored and supported.

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Q: Anti-retroviral therapy in children

Antiretroviral therapy (ART) in children is a critical aspect of managing HIV infection.

1. Initiation of ART:

- ART should be initiated in all HIV-infected children, regardless of CD4 count or clinical stage, as soon as possible after diagnosis.
- In infants diagnosed with HIV, ART should be started immediately, as early treatment significantly improves outcomes.

2. Regimen Selection:

- The choice of ART regimen depends on the child's age, weight, clinical condition, potential side effects, drug interactions, and resistance patterns.

- First-line regimens typically include two nucleoside reverse transcriptase inhibitors (NRTIs) and a non-nucleoside reverse transcriptase inhibitor (NNRTI) or an integrase strand transfer inhibitor (INSTI).

3. Dosage and Administration:

- Dosages of antiretroviral drugs are based on the child's weight and age. It's crucial to adjust dosages as the child grows.
- Some antiretroviral drugs are available in pediatric formulations, such as syrups or dispersible tablets, to facilitate administration in young children.

4. Monitoring and Adherence:

- Regular monitoring of viral load, CD4 count, and clinical status is essential to assess response to ART and make necessary adjustments.
- Adherence to ART is critical for its success. Poor adherence can lead to virological failure and the development of drug resistance.

5. Management of Side Effects:

- Side effects vary depending on the drugs used. Common side effects include gastrointestinal disturbances, rash, and metabolic abnormalities.
- Monitoring for and managing side effects is important to maintain adherence and quality of life.

6. Drug Resistance:

- In cases of virological failure, resistance testing should be considered to guide the selection of a new regimen.
- Switching to a second-line or third-line regimen may be necessary if resistance develops.

7. Special Considerations:

- In children with tuberculosis (TB), careful consideration is needed due to drug interactions between antiretroviral drugs and TB medications.
- In adolescents, issues of privacy, independence, and transitioning to adult care need to be addressed.

8. Prevention of Opportunistic Infections:

- In addition to ART, prophylaxis against opportunistic infections, such as *Pneumocystis jirovecii* pneumonia (PCP), may be indicated based on the child's age and immune status.

Drug Class	Drug Name	Mechanism of Action	Pediatric Dose	Common Side Effects
Nucleoside Reverse Transcriptase Inhibitors (NRTIs)	Zidovudine (AZT)	Inhibits reverse transcriptase by incorporating into viral DNA	180-240 mg/m ² BID	Anemia, neutropenia, lactic acidosis, myopathy
NRTIs	Lamivudine (3TC)	Inhibits reverse transcriptase by terminating DNA chain	4 mg/kg BID (max 150 mg BID)	Headache, fatigue, nausea, pancreatitis
NRTIs	Abacavir (ABC)	Inhibits reverse transcriptase by terminating DNA chain	8 mg/kg BID (max 300 mg BID)	Hypersensitivity reaction, nausea, headache
Non-Nucleoside Reverse Transcriptase Inhibitors (NNRTIs)	Nevirapine (NVP)	Binds directly to reverse transcriptase and inhibits RNA-dependent DNA polymerase	150-200 mg/m ² BID	Rash, hepatotoxicity, Stevens-Johnson syndrome
NNRTIs	Efavirenz (EFV)	Binds directly to reverse transcriptase and inhibits RNA-dependent DNA polymerase	200-600 mg once daily (based on weight)	CNS effects (dizziness, insomnia), rash

Protease Inhibitors (PIs)	Lopinavir/Ritonavir (LPV/r)	Inhibits HIV protease, preventing viral maturation	230/57.5 mg/m ² BID	Diarrhea, hyperlipidemia, insulin resistance
PIs	Atazanavir (ATV)	Inhibits HIV protease, preventing viral maturation	200-400 mg once daily (based on weight)	Hyperbilirubinemia, nausea, rash
Integrase Strand Transfer Inhibitors (INSTIs)	Raltegravir (RAL)	Inhibits HIV integrase, preventing integration of viral DNA into host genome	6 mg/kg BID (max 400 mg BID)	Insomnia, headache, increased creatine kinase
INSTIs	Dolutegravir (DTG)	Inhibits HIV integrase, preventing integration of viral DNA into host genome	1-2 mg/kg once daily (max 50 mg)	Insomnia, headache, hypersensitivity reaction
Entry Inhibitors	Enfuvirtide (T-20)	Inhibits fusion of HIV with host cell membrane	2 mg/kg BID (max 90 mg BID)	Injection site reactions, hypersensitivity, pneumonia
CCR5 Antagonists	Maraviroc (MVC)	Blocks CCR5 co-receptor on CD4 cells, preventing viral entry	Dose based on weight and concomitant medications	Cough, pyrexia, upper respiratory tract infections

- Some drugs require dose adjustments based on age, weight, and renal or hepatic function.
- Before initiating Abacavir, genetic testing for HLA-B*5701 should be done to assess the risk of hypersensitivity reaction.

- The choice of ART regimen should be individualized based on resistance patterns, potential drug interactions, and other patient-specific factors.

Regimens of antiretroviral therapy (ART) followed in India for children, based on the National AIDS Control Organisation (NACO) guidelines:

Age Group	First-Line Regimen	Second-Line Regimen	Third-Line Regimen (Salvage Therapy)
<3 years	Zidovudine (AZT) or Abacavir (ABC) + Lamivudine (3TC) + Lopinavir/Ritonavir (LPV/r)	Abacavir (ABC) + Lamivudine (3TC) + Atazanavir/Ritonavir (ATV/r) or Darunavir/Ritonavir (DRV/r)	Individualized based on resistance testing and available options. May include newer drugs like Dolutegravir (DTG), Raltegravir (RAL), or Etravirine (ETR).
3-10 years	Zidovudine (AZT) or Abacavir (ABC) + Lamivudine (3TC) + Efavirenz (EFV) or Nevirapine (NVP)	Abacavir (ABC) + Lamivudine (3TC) + Atazanavir/Ritonavir (ATV/r) or Darunavir/Ritonavir (DRV/r)	Individualized based on resistance testing and available options. May include newer drugs like Dolutegravir (DTG), Raltegravir (RAL), or Etravirine (ETR).
>10 years or >35 kg	Tenofovir (TDF) + Lamivudine (3TC) or Emtricitabine (FTC) + Efavirenz (EFV)	Zidovudine (AZT) + Lamivudine (3TC) + Atazanavir/Ritonavir (ATV/r) or Darunavir/Ritonavir (DRV/r)	Individualized based on resistance testing and available options. May include newer drugs like Dolutegravir (DTG), Raltegravir (RAL), or Etravirine (ETR).

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Q: Dry blood PCR for HIV/AIDS testing

Dry blood spot (DBS) PCR (Polymerase Chain Reaction) testing for HIV/AIDS is a method used for the detection of HIV genetic material (RNA or DNA) in dried blood samples. This method is particularly useful in resource-limited settings or for populations where venous blood draw may be challenging, such as in infants or in remote areas.

1. **Sample Collection:** A small amount of blood is collected via a finger prick or heel prick (in infants) and is then applied to a special filter paper card. The blood is allowed to dry completely on the paper.
2. **Transport and Storage:** The dried blood spot (DBS) cards can be easily transported to the laboratory without the need for refrigeration, making it a practical option for remote locations.
3. **Extraction of Genetic Material:** In the laboratory, a small portion of the dried blood spot is cut out. The genetic material (DNA or RNA) is then extracted from the cells in the blood spot.
4. **PCR Testing:** The extracted genetic material is subjected to PCR testing. This involves amplifying specific segments of the HIV genetic material to detectable levels. There are two types of PCR tests commonly used:
 - **HIV DNA PCR:** Used primarily for early infant diagnosis, as it can detect the presence of HIV DNA integrated into the host cells.
 - **HIV RNA PCR (Viral Load Testing):** Measures the amount of HIV RNA (viral load) in the blood, which is crucial for monitoring treatment effectiveness.
5. **Advantages:**
 - **Accessibility:** Facilitates testing in remote or resource-limited settings.
 - **Early Infant Diagnosis:** Allows for the detection of HIV in infants born to HIV-positive mothers, where antibody tests are not reliable due to the presence of maternal antibodies.
 - **Stability:** DBS samples are stable at room temperature and can be transported without cold chain logistics.
 - **Minimally Invasive:** Requires only a small amount of blood, making it less invasive, especially for infants and children.
6. **Limitations:**
 - **Sensitivity and Specificity:** While highly accurate, the sensitivity and specificity can vary based on the assay used and the quality of the sample.
 - **Requirement for Laboratory Processing:** Samples must be sent to a laboratory with the capacity to perform PCR testing.
7. **Interpretation of Results:** A positive result indicates the presence of HIV genetic material, confirming HIV infection. A negative result suggests the

absence of HIV, but timing of the test and potential exposure must be considered.

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Q: Diagnostic tests for HIV in neonates and children

Diagnosing HIV in neonates and children involves a combination of tests, primarily because the standard antibody tests used in adults are not reliable in infants due to the presence of maternal HIV antibodies.

1. Polymerase Chain Reaction (PCR) Tests:

- **HIV DNA PCR:** Detects the presence of HIV DNA integrated into the host cells. It is particularly useful for early infant diagnosis, as it can detect HIV infection in infants as young as 2-3 weeks old.
- **HIV RNA PCR (Viral Load Test):** Measures the amount of HIV RNA (viral load) in the blood. It can be used for early diagnosis and also for monitoring treatment effectiveness.

2. Antibody Tests:

- **ELISA (Enzyme-Linked Immunosorbent Assay):** Used for older children (usually over 18 months) to detect HIV antibodies. However, it's not reliable for infants due to maternal antibodies.
- **Western Blot:** Often used as a confirmatory test after a positive ELISA in older children.

3. Rapid HIV Antibody Tests:

- Used in older children for quick screening. These tests provide results within minutes but are subject to the same limitations as other antibody tests in infants.

4. p24 Antigen Test:

- Detects the p24 protein of HIV. It can be used in conjunction with antibody tests for diagnosis in older children but is less commonly used in neonates.

5. Complete Blood Count (CBC) and Differential:

- While not diagnostic for HIV, these tests can provide information on the child's immune status and help identify complications like infections or anemia.

6. **CD4 Count:**

- Measures the number of CD4 T-cells in the blood. It's used to assess the immune function and guide treatment decisions, rather than for initial diagnosis.

7. **Drug Resistance Testing:**

- Performed in cases where there is suspicion of drug-resistant HIV strains, especially in children born to mothers with known resistance or treatment failure.

Timing of Testing in Neonates:

- **Birth to 2 Weeks:** HIV DNA or RNA PCR tests can be performed. Antibody tests are not reliable due to maternal antibodies.
- **2 Weeks to 4 Weeks:** HIV DNA or RNA PCR tests are recommended.
- **1 Month to 18 Months:** HIV DNA or RNA PCR tests are the primary diagnostic tools. Antibody tests may still be influenced by maternal antibodies.
- **After 18 Months:** Antibody tests (ELISA, Western Blot, or rapid tests) can be used reliably.

Interpretation of Results:

- **Positive PCR Test:** Indicates HIV infection. Confirmatory testing and immediate initiation of antiretroviral therapy are recommended.
- **Negative PCR Test:** Suggests the absence of HIV infection, but repeat testing may be necessary to confirm, especially if there was recent exposure.

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Q: Clinical features diagnosis and treatment of Kala azar

Kala-azar, also known as visceral leishmaniasis, is a severe parasitic disease caused by the protozoan *Leishmania donovani*.

Clinical Features:

1. **Onset:** Often insidious, with symptoms developing over weeks to months.
2. **Fever:** Prolonged, high-grade, and intermittent. Often worse in the evenings.
3. **Weight Loss:** Significant and progressive.

4. **Weakness and Fatigue:** Common due to anemia and the systemic nature of the infection.
5. **Splenomegaly:** Enlargement of the spleen is a hallmark feature, often massive.
6. **Hepatomegaly:** Enlargement of the liver is also common.
7. **Lymphadenopathy:** Swelling of lymph nodes may occur.
8. **Pancytopenia:** Reduction in all types of blood cells (red cells, white cells, and platelets).
9. **Hyperpigmentation:** Darkening of the skin, particularly on the hands, feet, abdomen, and face, giving rise to the name "Kala-azar" (Black Fever).
10. **Other Symptoms:** May include abdominal discomfort, cough, diarrhea, and secondary infections due to immunosuppression.

Diagnosis:

1. **Clinical Assessment:** Based on symptoms, history of exposure in endemic areas, and physical examination findings (splenomegaly, hepatomegaly).
2. **Laboratory Tests:**
 - **Complete Blood Count (CBC):** Often shows pancytopenia.
 - **Bone Marrow Aspiration or Biopsy:** Demonstration of *Leishmania* amastigotes (LD bodies) in bone marrow smears is definitive.
 - **Splenomegaly Aspiration:** May be done for parasite detection but has a risk of bleeding.
3. **Serological Tests:**
 - **rK39 Rapid Diagnostic Test:** A rapid immunochromatographic test detecting antibodies against *Leishmania*.
 - **Direct Agglutination Test (DAT):** Detects antibodies and is highly sensitive.
4. **Molecular Tests:**
 - **Polymerase Chain Reaction (PCR):** Highly sensitive and specific but not widely available in all settings.
5. **Other Tests:** Liver function tests, renal function tests, and tests to rule out other causes of fever and splenomegaly.

Treatment:

1. **Antiparasitic Drugs:**

- **Liposomal Amphotericin B:** Preferred treatment in many regions due to its efficacy and safety profile.
 - **Miltefosine:** An oral agent effective against visceral leishmaniasis.
 - **Pentavalent Antimonials:** Such as sodium stibogluconate or meglumine antimoniate. Historically the mainstay of treatment but resistance is an issue in some areas.
 - **Paromomycin:** An aminoglycoside antibiotic with antileishmanial activity.
2. **Treatment Duration and Regimen:** Varies based on the drug used, patient's age, weight, and local guidelines.
 3. **Supportive Care:** Management of anemia, nutritional support, and treatment of secondary infections.
 4. **Monitoring:** Regular follow-up to monitor response to treatment and detect any relapse.
 5. **Prevention and Control:** Vector control (reducing sandfly populations), personal protective measures, and early diagnosis and treatment of cases in endemic areas.

Special Considerations:

- **Pregnancy:** Certain drugs like miltefosine are contraindicated.
- **Children:** Dosage adjustments are necessary.
- **Immunocompromised Patients:** May have atypical presentations and require longer treatment.

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Q: Malaria clinical features and treatment

Aspect	Details
Causative Organism	<i>Plasmodium falciparum</i> , <i>P. vivax</i> , <i>P. ovale</i> , <i>P. malariae</i> , and <i>P. knowlesi</i> .
Species	<i>P. falciparum</i> : Most severe, can cause cerebral malaria. <i>P. vivax</i> : Common, can cause relapses. <i>P. ovale</i> : Similar to <i>P. vivax</i> , less common.

	<i>P. malariae</i> : Can cause chronic infection. <i>P. knowlesi</i> : Zoonotic, can be severe.
Clinical Features	Fever: Often cyclical, corresponding to parasite life cycle 48 – 72hr. Chills and Sweating: Classic malarial symptoms. Headache, Muscle Aches, Fatigue. Vomiting, Diarrhea, Abdominal Pain. Anemia, Jaundice (in severe cases). Splenomegaly, Hepatomegaly. In <i>P. falciparum</i> : Cerebral malaria, severe anemia, respiratory distress, renal failure.
Diagnosis	Microscopy : Gold standard. Thick and thin blood smears. Rapid Diagnostic Tests (RDTs) : Detect antigens, useful where microscopy is not available. Molecular Tests : PCR for species identification and drug resistance. Complete Blood Count (CBC) : May show anemia, thrombocytopenia. Liver and Renal Function Tests : In severe cases.
Treatment	Artemisinin-based Combination Therapies (ACTs) : First-line for <i>P. falciparum</i> . Chloroquine : For <i>P. vivax</i> , <i>P. ovale</i> , <i>P. malariae</i> (if sensitive). Primaquine : For radical cure of <i>P. vivax</i> and <i>P. ovale</i> (to prevent relapses). Quinine or Quinidine : In severe cases or when ACTs are not available. Supportive Care : Management of complications like anemia, dehydration, seizures. Prevention : Mosquito control, prophylaxis in high-risk areas, use of bed nets.

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Q: Define drug resistant malaria, what are the different types of drug resistance as per WHO criteria. Discuss the various management strategies of Drug resistant Malaria

Definition of Drug-Resistant Malaria:

Drug-resistant malaria refers to malaria infections that are less responsive or unresponsive to standard antimalarial drugs. This resistance occurs when malaria parasites evolve and develop mechanisms to survive exposure to the drugs that were previously effective against them.

Types of Drug Resistance as per WHO Criteria:

1. **Chloroquine Resistance:** Resistance to chloroquine, historically the mainstay of malaria treatment, particularly in *Plasmodium falciparum*.
2. **Sulfadoxine-Pyrimethamine Resistance:** Resistance to sulfadoxine-pyrimethamine, often used in intermittent preventive treatment.
3. **Artemisinin Resistance:** Reduced susceptibility of *P. falciparum* to artemisinin derivatives, characterized by delayed parasite clearance.
4. **Multidrug Resistance:** Resistance to multiple antimalarial drugs, making treatment more challenging.
5. **Partner Drug Resistance:** Resistance to the partner drugs used in combination with artemisinin derivatives in artemisinin-based combination therapies (ACTs).

Management Strategies of Drug-Resistant Malaria:

1. **Use of Combination Therapies:** Using ACTs (artemisinin-based combination therapies) to reduce the risk of resistance development.
2. **Switching Drug Regimens:** Changing to alternative medications or combinations when resistance is detected.
3. **Monitoring Drug Efficacy:** Regular surveillance to monitor the efficacy of antimalarial drugs and detect resistance early.
4. **Tailoring Treatment to Resistance Patterns:** Adapting treatment guidelines based on local resistance patterns and WHO recommendations.
5. **Prevention of Transmission:** Vector control measures to reduce malaria transmission, thereby decreasing the overall burden and spread of resistant strains.
6. **Education and Awareness:** Educating healthcare providers and communities about the importance of completing treatment courses and avoiding misuse of antimalarial drugs.
7. **Research and Development:** Investing in research to develop new antimalarial drugs and combination therapies.
8. **International Collaboration:** Collaborating across borders to monitor and manage drug resistance, as resistant strains can spread from one region to another.
9. **Treatment of Complicated Malaria:** In cases of severe or complicated malaria, especially with drug-resistant strains, hospitalization and intensive supportive care may be necessary.

10. **Prophylactic Measures:** In areas with known drug resistance, appropriate prophylactic measures and travel advice for those at risk.

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Q: Malaria prophylaxis

Malaria prophylaxis involves taking medication to prevent malaria infection, particularly when traveling to areas where the disease is endemic. The choice of prophylactic medication depends on several factors, including the destination, the traveler's health status, and the local patterns of drug resistance.

1. Risk Assessment:

- Determine the risk of malaria in the destination area.
- Consider the traveler's health status, age, pregnancy status, and any drug allergies.

2. Choice of Prophylactic Medication:

- **Chloroquine:** Used in areas without chloroquine-resistant *P. falciparum*.
- **Atovaquone-Proguanil (Malarone):** Effective in most areas, including those with drug resistance.
- **Doxycycline:** An alternative, especially in areas with multidrug resistance.
- **Mefloquine:** Used in areas with chloroquine-resistant *P. falciparum*, but has some neuropsychiatric side effects.
- **Primaquine:** Effective against *P. vivax* and for radical cure of liver stages.

3. Timing of Prophylaxis:

- Start prophylaxis before entering the malaria-endemic area (usually 1-2 weeks prior).
- Continue during the stay.
- Continue after leaving the area (usually for 4 weeks, but duration varies with the drug).

4. Adherence to Medication:

- Strict adherence to the medication schedule is crucial for effectiveness.
- Do not skip doses.

5. Monitoring for Side Effects:

- Be aware of potential side effects and seek medical advice if they occur.

6. Non-Pharmacological Measures:

- Use insect repellent containing DEET.
- Sleep under insecticide-treated bed nets.
- Wear long sleeves and trousers, especially during peak mosquito activity times.
- Consider insecticide-treated clothing in high-risk areas.

7. Special Considerations:

- Pregnant women, children, and individuals with certain medical conditions may have specific prophylaxis needs.

8. Awareness of Limitations:

- No prophylactic regimen is 100% effective; be vigilant for symptoms of malaria.

Medication	Regimen & Dose	Side Effects
Chloroquine	Adult: 500 mg once weekly. Child: 8.3 mg/kg once weekly (max 500 mg). Start 1-2 weeks before travel, continue during stay, and for 4 weeks after leaving.	Gastrointestinal upset. Headache. Pruritus (especially in dark-skinned individuals). Rarely, visual disturbances and neuropsychiatric effects.
Atovaquone-Proguanil (Malarone)	Adult: 1 adult tablet daily. Child: Dose based on weight. Start 1-2 days before travel, continue during stay, and for 7 days after leaving.	Abdominal pain. Nausea, vomiting. Diarrhea. Headache. Rarely, mouth ulcers and hair loss.
Doxycycline	Adult: 100 mg daily. Child: ≥8 years, 2 mg/kg daily (max 100 mg). Start 1-2 days before travel, continue during stay, and for 4 weeks after leaving.	Photosensitivity. Gastrointestinal upset. Candidiasis (yeast infections). Dental discoloration in children <8 years. Contraindicated in pregnancy.

Mefloquine	Adult: 250 mg once weekly. Child: Dose based on weight. Start 2-3 weeks before travel, continue during stay, and for 4 weeks after leaving.	Neuropsychiatric effects (e.g., anxiety, hallucinations). Dizziness. Gastrointestinal upset. Headache. Contraindicated in those with a history of seizures or psychiatric disorders.
Primaquine	Adult: 30 mg daily. Child: Dose based on weight. Start 1-2 days before travel, continue during stay, and for 7 days after leaving.	Gastrointestinal upset. Hemolytic anemia in individuals with G6PD deficiency. Methemoglobinemia. Contraindicated in pregnancy and G6PD deficiency.

Q: What are management guidelines of malaria under the national programme?

The management guidelines under the Indian National Programme emphasize prompt diagnosis, appropriate treatment based on species and severity, prevention strategies, and surveillance.

The management of malaria under the Indian National Programme follows the guidelines provided by the National Vector Borne Disease Control Programme (NVBDCP), which is a part of the Directorate General of Health Services, Ministry of Health & Family Welfare, Government of India.

1. Diagnosis:

- Prompt and accurate diagnosis is crucial.
- Use Rapid Diagnostic Tests (RDTs) and/or microscopy for confirmation.
- Microscopy remains the gold standard for diagnosis.

2. Treatment:

- Treatment should be based on the species of malaria and the severity of the disease.

1. *Plasmodium falciparum* Malaria:

- **First-line Treatment:**
 - **Artemisinin-based Combination Therapy (ACT)** is recommended.
 - The preferred combination is **Artesunate (AS) + Sulfadoxine-Pyrimethamine (SP)**.

- In areas with known resistance to SP, alternative ACTs like **Artemether-Lumefantrine (AL)** are recommended.
- **Dosage:**
 - The dosage of ACTs is based on the patient's weight and age.
 - Treatment is typically given for 3 days.
- **Severe Cases:**
 - Intravenous or intramuscular artesunate is the treatment of choice.
 - Followed by a full course of ACT once the patient can take oral medication.

2. *Plasmodium vivax* Malaria:

- **Chloroquine:**
 - Chloroquine is the first-line treatment for *P. vivax* malaria.
 - Typically administered over 3 days.
- **Radical Cure with Primaquine:**
 - To prevent relapse, primaquine is given for 14 days.
 - It's crucial to test for G6PD deficiency before administering primaquine due to the risk of hemolytic anemia.
- **Dosage:**
 - Dosage of chloroquine and primaquine is based on the patient's weight.

3. *Mixed Infections (P. falciparum and P. vivax)*:

- Treat as per *P. falciparum* malaria with ACT.
- Follow up with primaquine for radical cure of *P. vivax*, after testing for G6PD deficiency.

4. *Severe Malaria*:

- **Hospitalization:**
 - Patients with severe malaria should be hospitalized immediately.
- **Intravenous or Intramuscular Artesunate:**
 - This is the treatment of choice for severe malaria.

- Administered for at least 24 hours or until the patient can tolerate oral medication.
- **Transition to Oral Therapy:**
 - Once the patient is stable and can tolerate oral medication, switch to a full course of ACT.

4. Prevention and Control:

- Use of Long-Lasting Insecticidal Nets (LLINs) or Indoor Residual Spraying (IRS).
- Larval control measures.
- Prompt case detection and treatment.
- Awareness and education campaigns.

5. Monitoring and Surveillance:

- Regular monitoring of drug efficacy.
- Surveillance for early detection and containment of outbreaks.

6. Special Populations:

- Pregnant women: Avoid primaquine; use alternative regimens.
- Children: Dosing as per weight and age.

7. Drug Resistance:

- Monitor for signs of drug resistance.
- Adapt treatment policies based on resistance patterns.

8. Reporting:

- Mandatory reporting of all malaria cases to the NVBDCP.

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Q: Enumerate manifestations of Severe Malaria and their management in table

Manifestations of Severe Malaria	Management
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Cerebral Malaria (Altered consciousness, seizures, coma)	Immediate hospitalization. Intravenous or intramuscular artesunate. Supportive care: seizure management, maintaining airway and breathing. Close monitoring of neurological status.
Severe Anemia (Hemoglobin <5 g/dL)	Blood transfusion if indicated. Intravenous or intramuscular artesunate. Monitor hemoglobin levels and vital signs.
Respiratory Distress (Acidotic breathing, pulmonary edema)	Oxygen therapy. Intravenous or intramuscular artesunate. Supportive care: fluid management, monitoring oxygen saturation. Mechanical ventilation if necessary.
Acute Kidney Injury (Oliguria, elevated creatinine)	Intravenous or intramuscular artesunate. Fluid management, avoiding fluid overload. Monitor urine output and renal function. Dialysis if indicated.
Hypoglycemia (Blood glucose <40 mg/dL)	Intravenous dextrose. Regular monitoring of blood glucose levels. Intravenous or intramuscular artesunate.
Metabolic Acidosis	Intravenous bicarbonate if indicated. Monitor acidbase balance. Intravenous or intramuscular artesunate. Supportive care and fluid management.
Shock (Hypotension, poor perfusion)	Intravenous fluids. Vasopressors if necessary. Intravenous or intramuscular artesunate. Monitor hemodynamic status.
Spontaneous Bleeding (Disseminated intravascular coagulation)	Blood products (platelets, fresh frozen plasma) if indicated. Intravenous or intramuscular artesunate. Monitor coagulation profile.
Hyperparasitemia (>5% of red blood cells infected)	Intravenous or intramuscular artesunate. Consider exchange transfusion in extreme cases. Close monitoring of parasitemia levels.

Jaundice (Elevated bilirubin, liver enzymes)	Intravenous or intramuscular artesunate. Supportive care: monitoring liver function. Avoid hepatotoxic drugs.
Pulmonary Edema	Oxygen therapy. Intravenous or intramuscular artesunate. Diuretics if fluid overload is present. Mechanical ventilation if necessary.
Repeated Convulsions	Anticonvulsants as needed. Intravenous or intramuscular artesunate. Close neurological monitoring.

Artesunate is the drug of choice

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Q: Complications of severe falciparum malaria.

- ❖ The management of severe falciparum malaria and its complications requires a multi-faceted approach, including prompt antimalarial therapy, supportive care tailored to the specific complications, and close monitoring.
- ❖ Early recognition and aggressive management are key to improving outcomes and reducing mortality.
- ❖ Collaboration with critical care specialists, nephrologists, and other relevant specialists is often necessary for optimal management.

Major complications:

1. **Cerebral Malaria:**

- **Pathophysiology:** Parasitized red blood cells (RBCs) adhere to cerebral endothelium, causing obstruction, inflammation, and endothelial damage. This leads to cerebral hypoxia, edema, and potentially herniation.
- **Clinical Presentation:** Altered mental status ranging from confusion to coma, seizures, often accompanied by fever.
- **Management:** Immediate antimalarial therapy (e.g., intravenous artesunate), supportive care, management of raised intracranial pressure, seizure control, and close monitoring of neurological status.

2. **Severe Anemia:**

- **Mechanism:** Hemolysis of parasitized RBCs, dyserythropoiesis, and splenic sequestration.
- **Clinical Features:** Pallor, fatigue, weakness, and in severe cases, high-output cardiac failure.
- **Management:** Blood transfusion in cases of severe anemia, antimalarial treatment, and supportive care.

3. **Acute Respiratory Distress Syndrome (ARDS):**

- **Pathogenesis:** Inflammatory cytokines and sequestered RBCs in pulmonary microvasculature lead to increased capillary permeability, interstitial and alveolar edema.
- **Symptoms:** Severe dyspnea, hypoxemia, and respiratory distress.
- **Management:** Oxygen therapy, mechanical ventilation if necessary, and careful fluid management.

4. **Acute Kidney Injury (AKI):**

- **Etiology:** Ischemia due to hypovolemia, hemoglobinuria, and direct parasitic effects on renal tubules.
- **Presentation:** Reduced urine output, elevated creatinine and urea, fluid overload.
- **Management:** Fluid resuscitation, renal replacement therapy (dialysis) if indicated, and antimalarial therapy.

5. **Hypoglycemia:**

- **Causes:** Quinine-induced hyperinsulinemia, impaired gluconeogenesis, and increased glucose consumption by the host and parasite.
- **Clinical Importance:** Particularly dangerous in children and pregnant women; can lead to seizures and coma.
- **Management:** Frequent blood glucose monitoring, intravenous glucose administration.

6. **Metabolic Acidosis:**

- **Mechanism:** Lactic acidosis from anaerobic glycolysis due to impaired tissue perfusion, renal dysfunction.
- **Consequences:** Can lead to shock and multi-organ failure.

- **Management:** Correction of underlying causes, administration of IV fluids, and bicarbonate therapy if indicated.

7. **Disseminated Intravascular Coagulation (DIC):**

- **Pathophysiology:** Activation of coagulation pathways, leading to microthrombi formation and consumption of clotting factors and platelets.
- **Clinical Manifestations:** Spontaneous bleeding, petechiae, and prolonged clotting times.
- **Management:** Treatment of the underlying infection, supportive care, transfusions if necessary.

8. **Hepatic Dysfunction:**

- **Presentation:** Jaundice, hepatomegaly, elevated liver enzymes.
- **Implications:** Can affect drug metabolism and contribute to hypoglycemia.
- **Management:** Supportive care, monitoring liver function, and adjusting medication dosages as needed.

9. **Splenic Rupture:**

- **Risk Factors:** Hyperactive spleen due to increased phagocytic activity.
- **Symptoms:** Sudden abdominal pain, hemodynamic instability.
- **Management:** Surgical intervention in case of rupture, conservative management in case of enlargement without rupture.

10. **Cardiovascular Collapse:**

- **Due to:** Severe dehydration, shock, acidosis, and cardiac dysfunction.
- **Result:** Hypotension, arrhythmias, reduced cardiac output.
- **Management:** Fluid resuscitation, vasopressors, inotropic support.

11. **Blackwater Fever:**

- **Characteristics:** Intravascular hemolysis, hemoglobinuria, renal failure, and jaundice.
- **Associated with:** Repeated malaria infections, certain antimalarial drugs.